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A CLINICAL ANALYSIS OF PRIMARY ATYPICAL PNEUMONIA WITH A DISCUSSION OF ELECTROCARDIOGRAPHIC FINDINGS *

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DURING the last few years, numerous reports have appeared in medical literature describing a type of pneumonia differing from both lobar and bronchopneumonia. Many confusing terms and designations have been suggested for this disease but it is generally referred to as atypical pneumonia.

Although the disease is commonly regarded as a new entity, Dingle and Finland,[1] in reviewing the literature on this type of pneumonia, found that as long ago as 1872, a similar disease and pathological picture was described. They noted also the existence of a condition in the military forces, even before the pandemic of influenza in 1918, called by such names as "soldier's pneumonia" and "catarrhal fever." It seems apparent even from this meager information that the disease entity is probably not new at all, but merely has escaped recognition because of the inability of physicians fully to utilize the roentgen-ray in their study of obscure respiratory infection.

Efforts to determine a common causative agent of this disease process have not met with much success. Isolated cases and even small groups of cases have been shown to be caused by various filterable viruses, such as influenza A and B, psittacosis, ornithosis and the virus of lymphocytic choriomeningitis. Atypical pneumonic lesions have been observed in several of the rickettsial diseases, namely, typhus, Rocky Mountain spotted fever and both the Australian and American Q fever.[2] The clinical picture of pulmonary coccidioidomycosis, a fungus disease, not infrequently resembles that of atypical pneumonia. The protozoan, toxoplasma [3] has been reported to produce an atypical pneumonic process. Finland [4] has stated that the pneumococcus and even the streptococcus may produce atypical pulmonary processes, particularly in children.

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In all probability, this disease is not a single entity, but a syndrome produced by a variety of agents, both non-bacterial and bacterial.

Even after all attempts to determine an etiologic agent have been exhausted, there still is an appreciable number of cases in which the clinical and roentgenographic picture is that of atypical pneumonia. This condition, which has been rather prominent in military installations [5, 6, 7, 8, 9, 10] has been designated by the Surgeon General as primary atypical pneumonia, etiology unknown.

It is with the latter group that this paper is concerned. During the period from May, 1943 to April, 1944, 678 cases were admitted to the Regional Station Hospital, Greensboro, North Carolina, with the diagnosis of pneumonia. Of this number, 187 were considered to be lobar pneumonia of pneumococcic origin. The remainder, or 491, were classified as atypical in character.

This present analysis is based on the clinical experience with 321 of these cases, in which the etiology was unknown or undetermined. Inasmuch as laboratory facilities were not available for extensive virus studies, it is possible that some cases may have been due to such an agent, but with the evidence at hand they had to be regarded as of unknown etiology. The question might arise as to whether some of these cases were not of bacterial origin, as has been suggested by Finland.[4] Any answer to that problem is difficult, but sputum studies and blood cultures failed to corroborate this possibility. The latter will be discussed in detail later.

The establishment of absolute diagnostic criteria in this disease is impossible. However, the following points were considered before the case was designated as one of primary atypical pneumonia:

1. Inability to determine a known etiological agent.
2. History of gradual onset.
3. Benign but prolonged course.
4. Minimal physical findings.
5. Roentgenographic evidence of mottled infiltration usually lacking the marked homogeneous density of lobar pneumonia.
6. An essentially normal initial leukocyte count.
7. In general, poor response to sulfonamide therapy.

Obviously, strict adherence to all of these criteria was not feasible, so that each case was considered individually. In some of the cases, the diagnosis had to be made by exclusion.

Age, Sex, and Race Distribution. Chart 1 illustrates the relative frequency of the various age groups. In this series those aged 18 predominated, approximately 30 per cent falling into this group. Eighty per cent of the cases occurred between the ages of 18 and 26, which would be expected in a post where Air Corps personnel were undergoing training. The frequent occurrence of this disease in young adults is confirmed by recent reports in the literature.[1, 2, 4, 5, 6, 7, 8, 9, 11] All of these investigations were made in either

military posts or civilian schools, so that the age incidence would of necessity be limited to the younger age groups. However, it is notable that relatively little has been published concerning atypical pneumonia in the older age groups.

Inasmuch as only one case in this series was a female, no data on sex incidence are included.

In this series, 294 of the cases were white and 27 were colored. Although it was difficult to interpret these figures, owing to the varying

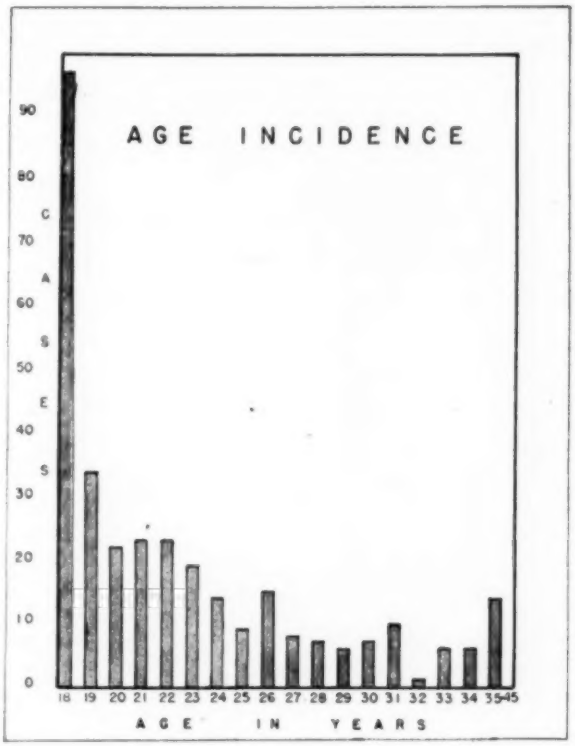

CHART 1.

monthly strengths of white and colored troops, it was our impression that the disease was not quite so prevalent in the colored as in the white race.

It was also thought that atypical pneumonia was more frequent in recruits than in soldiers who had finished their training; Chart 2 demonstrates the incidence of the disease according to the length of service. The highest incidence, 76.6 per cent, occurred during the first three months of service. This condition would be anticipated in a basic training center, where the majority of the personnel were trainees who remained at the post for only a short interval. Eighty-five and three-tenths per cent of the cases developed during the first six months of service, which suggests that this disease, at

least in some respects, resembles other "contagious" diseases which show a similarly high incidence during this period. In other words, the longer the soldier was in service, the less prone he was to develop atypical pneumonia. Of the 321 cases, only 16 had been in service in excess of one year. This group would be generally classified as permanent party personnel, who constituted about one-seventh of the entire post strength. On the basis of incidence for the entire post, this number should have been four times as many as actually occurred. In the main, the disease appeared most frequently in soldiers who had little military service. Similar observations relevant to the high incidence in recent inductees have been reported from other military installations.[6]

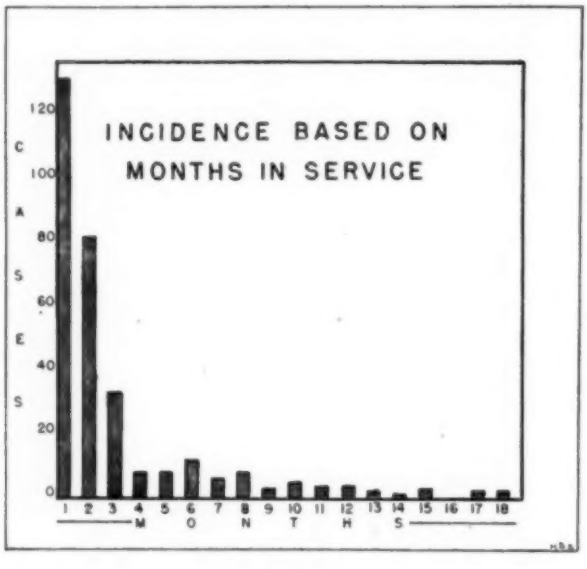

CHART 2.

Seasonal Incidence. Chart 3 portrays the seasonal incidence in months for both atypical and lobar pneumonia, contrasted with the post strength. If the number of cases per thousand troops is used as a basis, it is evident that March stands out as the month of greatest incidence for atypical pneumonia. The next in frequency were December, January, and February. Lobar pneumonia, on the other hand, attained its highest incidence in January and was less commonly observed during the month of March. A situation which prevailed in this portion of North Carolina does not necessarily represent the incidence that would be expected throughout the country as a whole. It is also apparent that primary atypical pneumonia occurred quite commonly throughout the entire year, whereas lobar pneumonia, though appearing sporadically in the summer months, was essentially a late fall and winter disease in this locality.

Origin of Cases by States. In an effort to determine whether soldiers transported from one state to another far removed from their native environment were more susceptible to this disease than if they remained in relative propinquity to their homes, a survey was made to determine the soldier's home state. It was found that most soldiers originally came from New York, Pennsylvania, Massachusetts, New Jersey, North Carolina, Connecticut and Georgia, in the order named. It was, therefore, our impression that although changes from one climate to another may be a factor

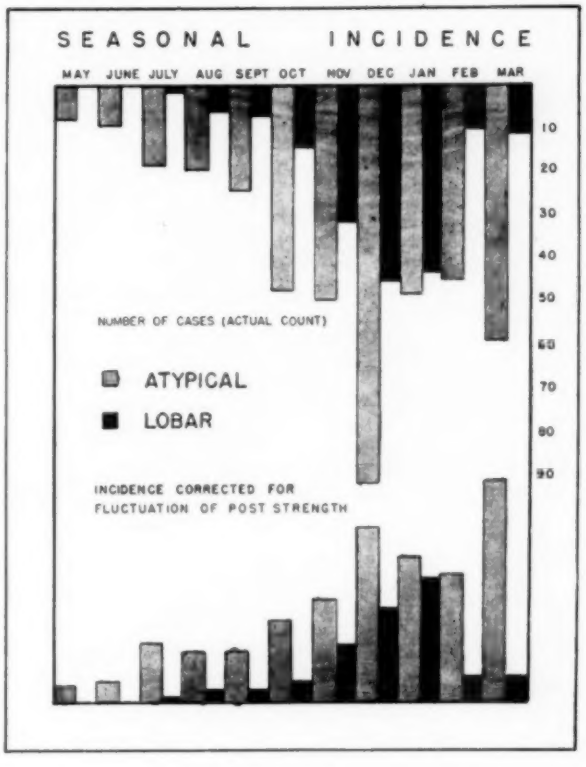

CHART 3.

in this disease, no statistical data were available as to the states which contributed the highest percentages to our training personnel. It is probable that the states which predominated in this series were those which contributed the greatest proportion of trainees to this post. In connection with this survey, it seemed expedient to ascertain whether the soldier had resided in a city or lived in a less congested area. In this series of 321 cases, 259, or 80 per cent, formerly resided in urban communities, whereas 62, or 20 per cent, were inhabitants of rural districts. It was our impression, based in part on the aforementioned figures, that primary atypical pneumonia occurred more frequently in city dwellers than in residents of

rural areas. This opinion again could not be verified, as comparative statistics were unavailable.

Past History. The past history was carefully investigated in each case. Particular attention was given to a history of frequent colds, sinusitis, tuberculosis and previous attacks of pneumonia. All cases were interrogated regarding a previous history of tonsillectomy and adenoidectomy.

Chart 4 reveals the pertinent information gleaned from the past history. Fifty-one cases, or 15.5 per cent, gave a history of prior attacks of pneumonia, but clinically this appeared to have no bearing on the outcome of the present disease. In only 29 cases, or 9 per cent, was there a history of frequent colds or sinusitis. A positive past history for tuberculosis was extremely rare (three cases). As might be expected, approximately 29

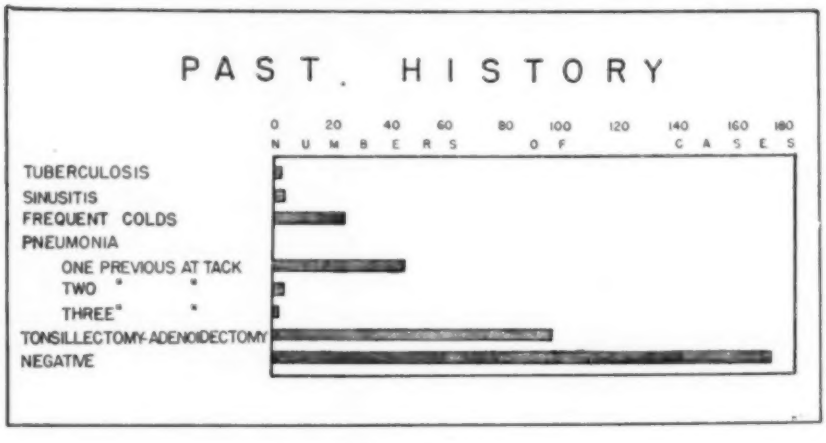

CHART 4.

per cent disclosed evidence of a previous tonsillectomy. An essentially negative history was elicited in 169 cases, representing 52.6 per cent of the series. A thorough survey of the past history exhibited no relation either to the incidence or severity of the disease.

Onset. The onset of primary atypical pneumonia has been variously described. Owen [8] noted a more sudden onset in the summer and a rather gradual onset in the winter months. Moore, Wightman, et al.[12] and Haight and Trolinger [9] observed a gradual onset in the majority of their patients. Correll and Cowan,[10] from their experience, classified the type of onset as sudden. Many factors have to be considered in evaluating the type of onset. Most of the symptoms existed for a considerable period preceding admission. A dry cough was present on an average of 10.3 days prior to admission. A productive cough, though uncommon, existed usually 9.5 days before admission. Sore throat antedated admission to the hospital by five days, pains in the chest by three days and fever by 2.5 days. Chills, which often denote a sudden type of onset, occurred in 98 cases, usually two days preceding admis-

sion. This latter symptom, which was evident in less than one-third of the cases, was the only one which suggested a sudden onset.

In view of the fact that practically all of the other symptoms were in evidence long before chills developed, the onset must be considered as gradual in type. This gradual onset occurred in most cases about 10 days before admission.

Symptoms. Chart 5 indicates the relative frequency of various symptoms which were present prior to admission. It should be stated that many of these symptoms persisted for varying lengths of time after hospitalization. The outstanding complaint, a dry cough, was present in 201 cases, or approximately two-thirds of the series. Fever was second in frequency and roughly in the same percentage. Malaise and chills occurred in about one-third of the cases. Upper respiratory infection, pain in the chest, and sore

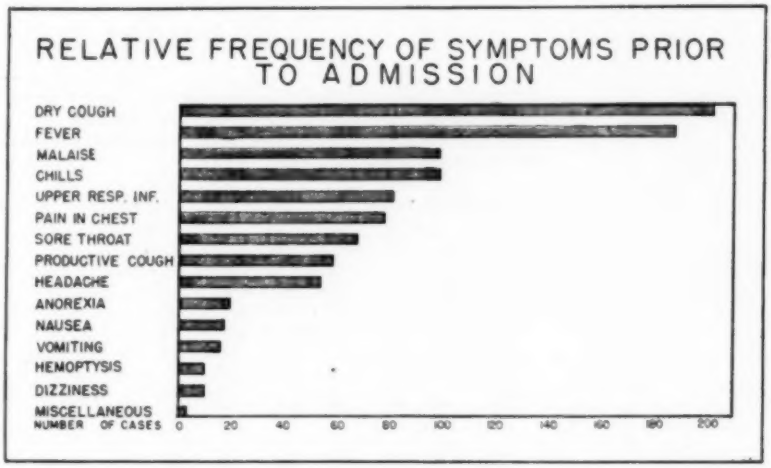

CHART 5.

throat were next in frequency and were experienced by about one-fourth of the cases. Productive cough was present in slightly less than one-fifth of the patients, another point differentiating atypical pneumonia from other types of pneumonia in which this symptom is usually present.

The relative percentages of some of these symptoms demand further mention. Chills were far more common (30.4 per cent) than one would expect, considering the usually benign course of the disease. Hemoptysis, a common occurrence in lobar pneumonia, was seen in only 3.4 per cent of the cases. About 25 per cent gave a history of recent or coexistent upper respiratory infection and the atypical pneumonic process was probably one and the same.

Appearance of Patient. Any appraisal of the degree of illness, based on the appearance upon admission is a matter of individual judgment, and is, obviously, not an exact index upon which to base conclusions. Observations

made on admission revealed that 174, or over half of the patients, were not considered acutely ill; 135 were regarded as moderately ill; and only 12 were deemed seriously, though not critically, ill. This latter group and some of those classified as moderately ill were the cases in which lobar pneumonia had to be excluded.

Weight. All patients were weighed either upon admission or shortly thereafter. Using standard weight tables as a basis, 47 per cent were found to be of normal weight and 53 per cent were equally divided between overweight and underweight. Subsequent observations revealed that weight bore no relation either to the development of the disease or to the severity of the ensuing pneumonic process.

Physical Findings. Some observers [7, 10] have reported a distinct paucity of physical findings in relation to the extent of the pathologic lesions as

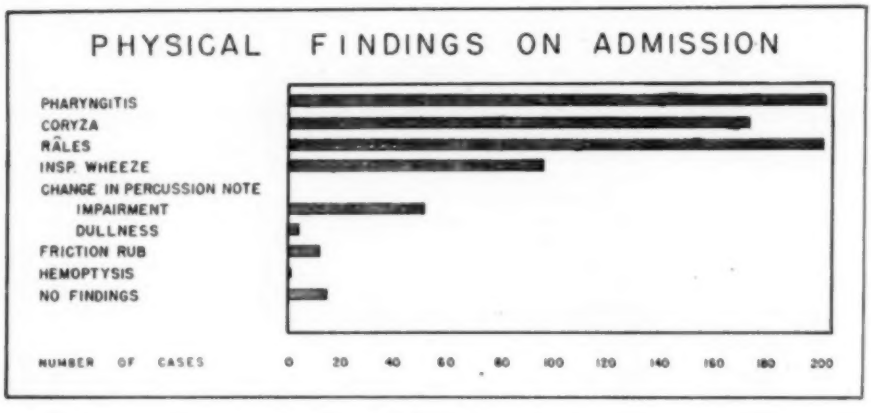

CHART 6.

demonstrated roentgenographically. Various writers have stated that early in the disease the majority of cases are characterized by the absence of clinical findings [1, 5, 13]; while others have remarked that the findings are minimal.[6, 14, 15]

In this series (chart 6) roughly two-thirds of the cases showed definite alterations from the normal physical findings. Fine or medium crackling râles were present over the affected area in 200 cases (60 per cent). Many were inaudible unless precipitated by coughing. In most of the cases these râles, though few in number, were present even on admission, if careful auscultation was carried out. Owen [8] reported that moist râles were the most consistent single finding in his series.

A most important physical finding, even in the absence of moist râles, was the evidence of a few high-pitched inspiratory wheezes, or sibilant râles, heard over the involved area. These wheezes were rather evanescent in character and often disappeared after a few deep inspirations, only to reappear a few hours later. This finding was observed in 29 per cent of the

cases. In view of the pathologic lesions of atypical pneumonia, this sign is of the utmost importance and has probably been overlooked by many clinicians who expected to elicit more conspicuous physical findings.

Changes in percussion note were present in relatively few cases, and this abnormality was termed simply impairment in resonance, since actual dullness was but rarely observed. This small percentage might be anticipated, as lobar distribution of the atypical process was an infrequent occurrence in this series.

Two hundred cases, or roughly two-thirds of the patients, had some degree of pharyngitis, varying from moderate injection to hyperemia. This finding was in accord with the clinical history (chart 5) which indicated

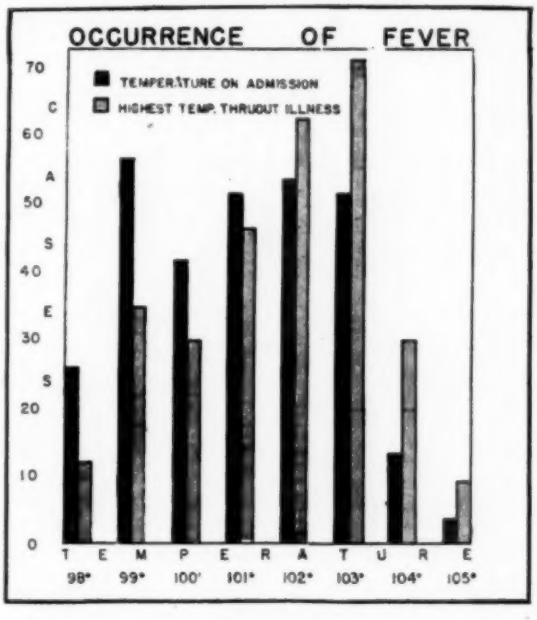

CHART 7.

that approximately that number of patients offered a complaint of sore throat or antecedent upper respiratory infection prior to admission.

Fever. Fever occurred in 90 per cent of the cases, whereas 10 per cent remained afebrile during their entire illness. As illustrated in chart 7, approximately 60 per cent of the cases varied between 101° and 103° F. A few cases were elevated to 104° and 105° F. This disease differs from lobar pneumonia in that a certain percentage enter the hospital with a normal temperature and develop a fever of 103° to 104° F., in 24 to 72 hours after admission.

Chart 8 graphically demonstrates the pulse rate upon admission and that encountered during the more acute stages of the disease. Seventy per cent of the cases showed a pulse rate that never exceeded 100 per minute. In

our series, atypical pneumonia was not characterized by a marked tachycardia either at the initial phase or during the peak of the illness. Tachycardia over 120 per minute and bradycardia were equally rare.

If it is recalled that in 60 per cent of the patients the temperature ranged between 101° and 103° F., it is obvious that the pulse rate was generally lower than might be expected with that degree of fever.

The same chart reveals that one-half of the patients in this series entered the hospital with a normal respiratory rate, and half of this group never exceeded this rate. Of the remainder, 45 per cent were admitted with a slight increase in respirations (22–24 per minute). During the peak of the

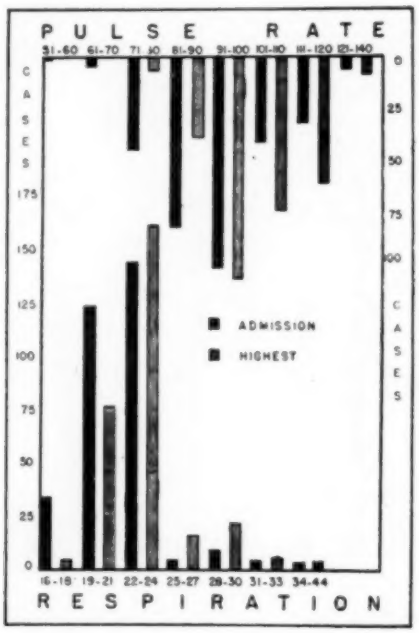

CHART 8.

illness, 50 per cent of the cases never developed a rate over 24 per minute. Unlike most other pneumonias, only 5.6 per cent, upon admission, were observed to have a respiratory rate of over 25 per minute, and only 14 per cent manifested hyperpnea during the height of the disease.

From these observations, it is apparent that in the majority of our cases the disease was accompanied by only a moderate elevation in pulse and respiratory rates.

Laboratory Examination. Blood studies were performed on 261 cases (84 per cent) during the initial stage of the disease. In only 10.3 per cent was there any evidence of anemia, as determined by hemoglobin estimations. In three-fourths of the cases (74 per cent), the hemoglobin determination

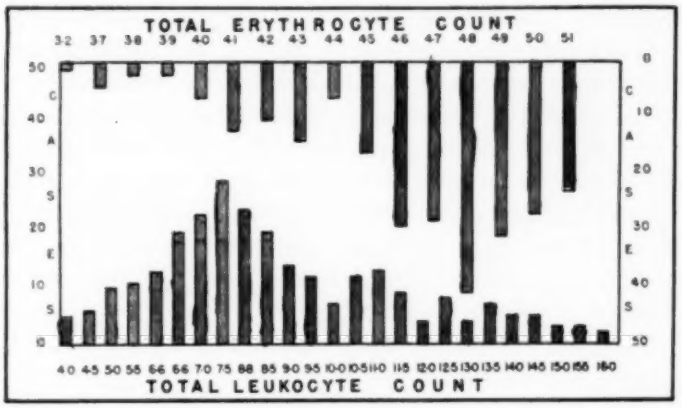

CHART 9.

was 85 per cent or more. Repeated hemoglobin estimations during the course of the disease showed no marked deviations from the initial level.

Chart 9 reveals that three-fourths of the cases had normal red cell counts (74 per cent), while only 4 per cent had counts of less than 4,000,000 per cu. mm. It is evident that there was no appreciable disparity between the hemoglobin determination and the erythrocyte count.

From the same chart, it is also apparent that the leukocyte count was normal (5,000–10,000) in most of the cases (68 per cent). A leukocytosis in excess of 10,000 per cu. mm. was present in 29 per cent, and in a small number of cases (4.4 per cent) the leukocyte count actually exceeded 15,000 per cu. mm. A leukopenia was found in only 4.4 per cent.

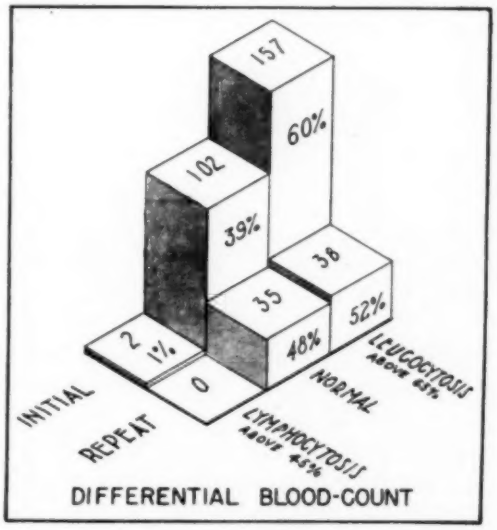

CHART 10.

From chart 10, differential counts revealed leukocytosis (over 65 per cent) in 60 per cent of the cases. A lymphocytosis (over 45 per cent) was found in only 1 per cent of the series; in the remaining 39 per cent the differential was normal. Subsequent differential counts on one-fourth of these cases revealed a persistent leukocytosis in 52 per cent. In our experience, the blood picture was one characterized by an essentially normal hemoglobin, red, and white cell count. It is also clear that the presence of leukocytosis, either absolute or relative, was no deterrent to the diagnosis of primary atypical pneumonia. A leukopenia or lymphocytosis was a rare occurrence.

Urinalyses were performed in all cases, and except for an occasional transitory febrile albuminuria, the findings were of no significance.

Blood cultures were taken in 53 cases and in all instances were reported negative.

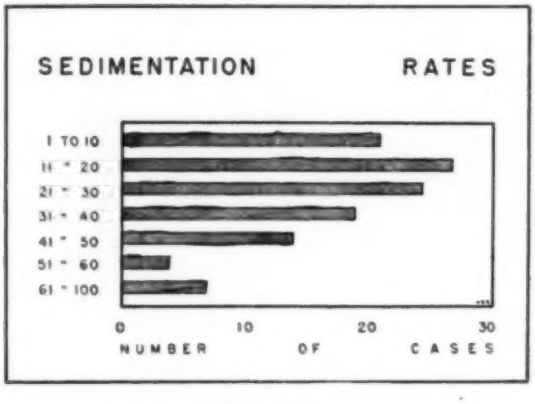

CHART 11.

Sedimentation rates were performed on somewhat more than one-third of the cases during the acute stage of the disease. Chart 11 indicates that more than one-third of the cases had a moderately accelerated rate (37.6 per cent), the remainder being almost evenly divided among a normal rate (18 per cent), a slightly elevated rate (23 per cent), and a rapid sedimentation rate (21.3). In the latter group were included those cases with rates in excess of 40 mm. per hour. In four of these cases, it exceeded 70 mm. per hour. Many of these rates were checked by the methods of Cutler, Wintrobe, and Westergren. Although this test was not used strictly as a guide in following a patient's progress, it was utilized as one of the criteria for discharge. In 170 cases, sedimentation rates were determined shortly before discharge, and in 154 of these cases, the rate was normal. In 16 cases the patients were discharged with a rate of 16 or above, and of this number, seven had a rate in excess of 20 mm. per hour and were discharged for emergency reasons. In our experience most of the cases had only a

moderately increased sedimentation rate, and this test was an aid in determining the optimum time for discharge. These observations tend to agree with those of van Ravenswaay,[6] who reported a moderate increase in the sedimentation rate in the initial stage of the disease. However, we were unable to corroborate his observation that this procedure served as an excellent guide to the progress of this disease, since in many instances, the sedimentation rate failed to parallel either the clinical or the roentgenographic findings.

Sputum. Sputum studies were done on the majority of cases. Fifty-one were examined particularly for evidence of *Mycobacterium tuberculosis*, but in no instances were acid-fast organisms observed.

In 135 cases, typing examinations were done to determine the presence of *Diplococcoccus pneumoniae*. In 117, pneumococci of no specific type were found, and in 18, type specific organisms were identified.

Table 1 indicates the predominant types.

TABLE I

Type I	2
Type III	3
Type IV	2
Type VII	1
Type VIII	1
Type XI	2
Type XIII	2
Type XVIII	1
Type XXII	1
Type XXVIII	1
Type XXIX	1
Type XXXIII	1
Number of cases with type specific organisms	18
Number of cases with "untypable" pneumococci	117
Total typings	135

The presence of various types of pneumococci in the sputum was difficult to evaluate. Types usually considered to be significant were found in the sputa of only 11 cases. These were Types I, III, IV, VII, VIII, and XI. The remaining seven sputa showed pneumococci of the higher numbered types. It is reasonable to assume that some of the 117 "untypable" pneumococci also belonged to the even higher numbered groups. It is also possible that these organisms may have been the cause of the pneumonic process, but when the clinical picture was considered, it was thought they were chiefly secondary invaders and any favorable results that might have occurred with sulfonamide therapy could be explained on this basis.

The question may arise as to whether some of the cases included in this series might be streptococcal in origin. The presence of pathogenic streptococci in the sputum is also difficult to evaluate. Although streptococci, staphylococci and *Micrococcus catarrhalis* were frequently found in the sputum, a pure culture of streptococci was obtained only in five instances. The benign clinical picture, the roentgenographic findings along with the total absence of complications, such as empyema, all were against this diagnosis. It is still possible that a few of these cases may have been unusual

streptococcal pneumonias, but such a diagnosis could not be substantiated by laboratory or clinical findings.

Roentgenographic Findings

A brief review of the pathology of atypical pneumonia is necessary before a proper evaluation of the roentgenographic changes can be made. Inasmuch as there were no fatalities in our series, original comment on the pathologic lesions of the disease cannot be offered. Relatively little has been published about the pathologic aspects of atypical pneumonia. Needles and Gilbert,[7] on one autopsy, noted on gross examination evidence of hilar node enlargement and a bronchial tree filled with creamy, viscid, yellow exudate which, when scraped away with difficulty, left a hemorrhagic bronchial mucosa. They also found hundreds of minute areas of infiltration resembling the gross picture of miliary tuberculosis, but the cut section was grayish pink, and scattered throughout the lung were nodules resembling bronchopneumonic consolidation. In summation, the picture was one of miliary pneumonitis, purulent bronchitis, and bronchiolitis. Microscopic examination revealed elongated, tortuous pulmonary alveoli lined with cuboidal epithelium. These cuboidal alveolar lining cells often projected into the bronchial lumina which were invariably filled with a purulent exudate essentially composed of polymorphonuclear elements. Fibrin was not noted. The interstitial tissues were greatly thickened, and the septa were packed with inflammatory cells of the round, wandering or plasma cell types. There was scattered loss of bronchial mucous membrane. The appearance was essentially one of bronchitis and interstitial pneumonia, and the pattern as a whole was that of proliferation and exudation.

Golden, quoted by Owen,[8] reported similar findings on gross examination, but stressed the point that both the peribronchial cellular infiltration and the associated congestion and edema of the lung tissue were essentially free of pus cells. Instead of the usual polymorphonuclear infiltration seen in pneumonias, these areas showed a predominance of lymphocytes, plasma cells and monocytes. If secondary infection occurred, hemorrhage, polymorphonuclear leukocytes, and fibrin formation became prominent, but failed to obscure the fundamental process.

Campbell et al.,[16] in their single necropsy, noted approximately the same findings as reported by the other investigators but observed evidence of atelectasis manifested by reduction of lung volume and decrease in the size of the alveoli.

Since the pathological sequence of events is probably a proliferative bronchitis, bronchiolitis, and interstitial pneumonitis, there must be a time when actual disease changes are present, though invisible by roentgen-ray. Showacre and his co-workers[17] concluded from their observations that an early film read as a negative does not rule out developing atypical pneumonia. When roentgenographic changes are evident, they may represent merely

endobronchial proliferation, peribronchial nodulations, or interstitial infiltration, or they may be the result of any combination of these processes.

The roentgen changes produced by this pulmonary reaction were multiform in character. These shadows often assumed the form of a veil-like extension projecting from the hilus, or were manifested by cloudy mottling or irregular areas of uneven density, varying in size from that of a pin point to nearly that of a dime. The typical lack of homogeneity of these shadows frequently made it difficult to delineate actual lobar involvement. Now and then, a dense form of consolidation not unlike that seen in lobar pneumonia was observed with distinct lobar demarcation.

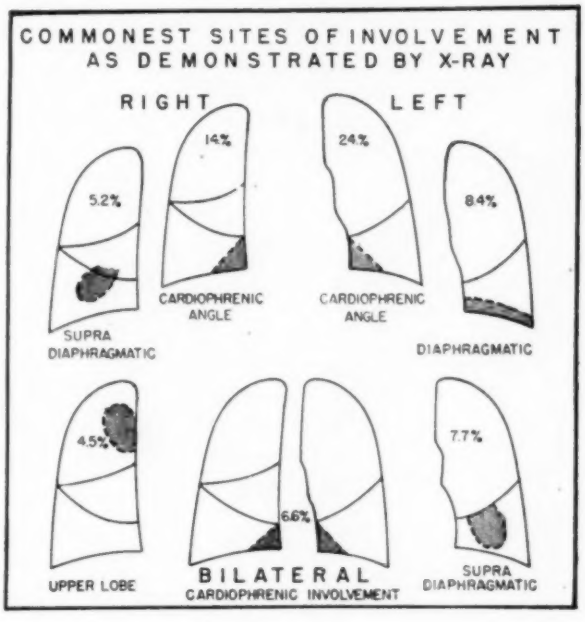

CHART 12.

In our survey, 17 more or less distinct areas of involvement were observed.

Roentgenograms illustrating the characteristic type of involvement in the six commonest sites are shown in figure 1.

Chart 12 portrays the pulmonary areas which were most frequently involved with an atypical pneumonic process. The appended table (2) reveals the areas of lessened incidence. It is apparent from chart 12 and table 2 that atypical pneumonia had a distinct predilection for involvement of the lower lung fields (79.9 per cent), and in this distribution, lesions of the dependent portion of the left lung predominated (41.6 per cent). It should also be pointed out that lesions of the left lung were far more common (48.1 per cent) than involvement of various regions of the right lung (34.8 per

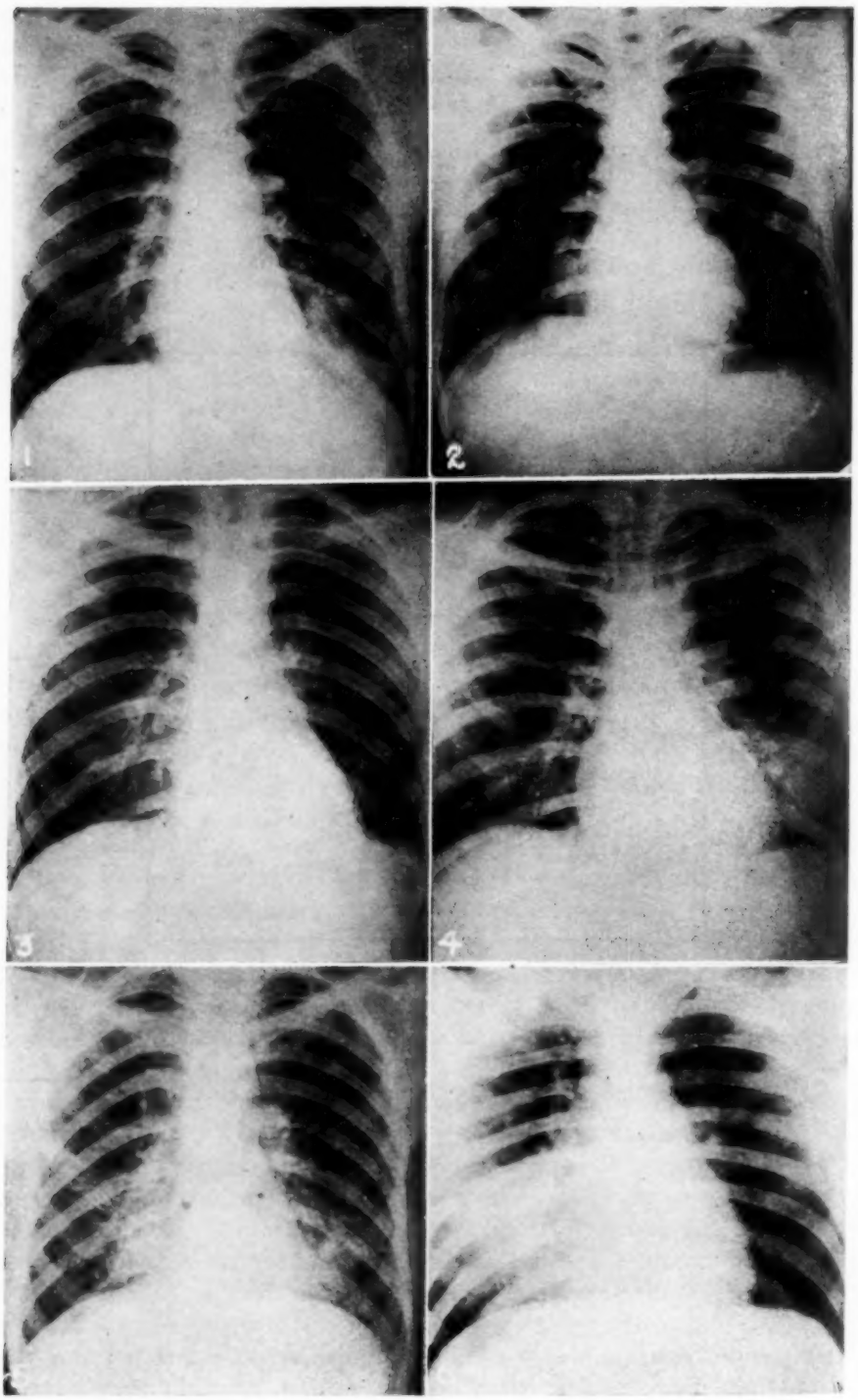

FIG. 1. (1) Left cardiophrenic lesion. (2) Right cardiophrenic lesion. (3) Left diaphragmatic involvement. (4) Left supradiaphragmatic lesion. (5) Bilateral cardiophrenic involvement. (6) Right supradiaphragmatic lesion.

cent). Bilateral processes were observed in only 12.6 per cent, and these were confined almost entirely to the lower lung fields.

Inasmuch as the pneumonic process only rarely involved an entire lobe, it seemed justifiable to use a more descriptive method in designating the site of involvement.

In considering pneumonias of the lower lung fields, it seemed more practical to designate the lesion according to its relationship to the cardiophrenic angles, the diaphragms, or the costophrenic angles. It should be pointed out that a cardiophrenic lesion may extend across the diaphragm to form a diaphragmatic lesion. All lesions of the lower lung field, not impinging directly upon the diaphragm and not distinctly lobar in distribution, were termed supradiaphragmatic. This designation was considered more accurate than any attempt to assign lobes in a poorly demarcated type of lesion.

In most of our cases, the lesion appeared to originate in the hilus and fan outward usually into the lower lung fields. When the lesion spread downward, it frequently extended along the cardiac borders or in a path posterior to the heart.

TABLE II

Less Frequent Sites of Involvement as Seen by Roentgen-Ray

Left hilar zone	3.9%
Right diaphragmatic region	3.4
Right costophrenic region	3.4
Bilateral diaphragmatic regions	3.2
Bilateral supradiaphragmatic regions	2.5
Left upper lobe	2.6
Right hilar zone	2.3
Left costophrenic region	1.5
Right middle lobe	1.5
Bilateral hilar zones	.3
Miscellaneous	4.5

It was also difficult at times to attach the proper term to those lesions emerging from the hilus and radiating towards or into the upper lobes, and to those confined essentially to the hilar region. In this latter category, only those cases were included in which the infiltration extended no further than the pulmonary midzones. By lobar involvement was meant those lesions which extended beyond the midzones into the periphery of the upper lobes (figure 2). It should be pointed out that the atypical process rarely occupied the entire lobe, and the density was seldom so marked as that seen in pneumococcic pneumonia. Furthermore, none of these areas was distinct in the same sense as that found in typical lobar consolidation.

Table 3 shows the incidence of atypical pneumonic involvement in our series, utilizing the aforementioned terminology, as compared with similar series using the lobar type of designation.

Roentgenographic findings disappeared on the average in about 18 days, although in seven cases, roentgenographic abnormalities persisted for 60 days or more. One case cleared in three days. In evaluating the time required for clearance of roentgenographic manifestations, consideration should be given to the history of the onset which in many cases antedated the

admission to the hospital by several days. Undoubtedly, if it were possible to roentgen-ray all cases at the onset of initial symptoms, the length of time required for clearance would be even longer than was actually determined. In other words, the time required for roentgen clearance is dependent on the stage of the disease upon which the original roentgenogram was taken.

It has been postulated that the diagnosis of atypical pneumonia is made by the roentgen-ray.[7, 13] In a corroborative sense, this statement is true, but it is also possible with the aid of a careful history, thorough physical examination, and proper evaluation of laboratory work, to make a reasonably

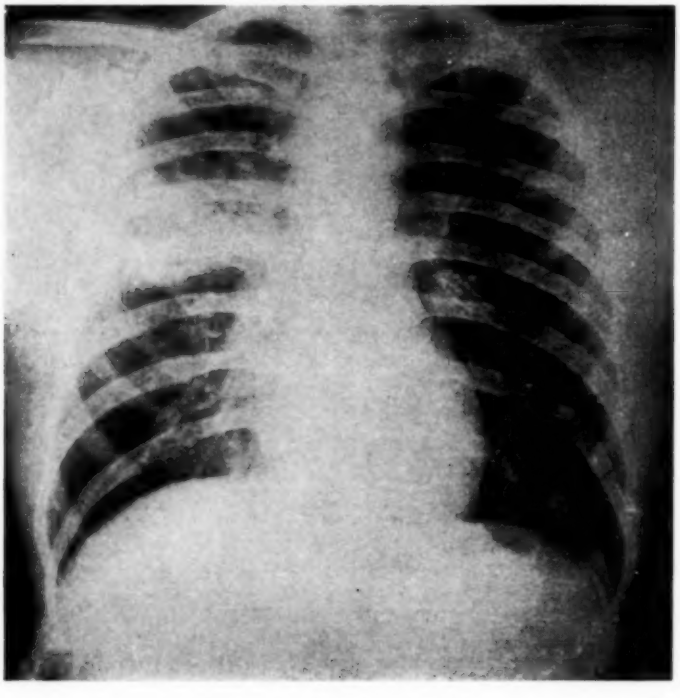

FIG. 2. Lobar involvement.

correct diagnosis without the aid of the roentgen-ray. It should be recalled that the roentgen findings, though relatively characteristic of this disease, are not pathognomonic, since other pathological processes, particularly pulmonary tuberculosis, may simulate the condition. It is an indisputable fact that the roentgen-ray is a most valuable adjunct, but the diagnosis should not be made solely by this method.

COURSE AND TREATMENT

The treatment of atypical pneumonia is governed by the particular phase of the disease with which the physician is dealing. The disease may be divided into three stages: acute, subsiding, and convalescent.

The acute stage was manifested by at least one of the following findings: persistent fever, abnormal chest signs, characteristic roentgenogram, or the presence of toxemia. Once this stage had been determined, the therapy was either symptomatic or chemotherapeutic. Bed rest was essential at this time. A soft bland diet was routinely prescribed and efforts were made to maintain a fluid intake of at least 3000 c.c. per day. The common expectorants were ordinarily indicated and steam inhalations often afforded relief. Symptoms resulting from pharyngitis and coryza responded to the usual remedies. Antipyretic drugs were seldom utilized.

TABLE III

	Dingle[15]	Owen[8]	Wightman[12] Showacre Moore	Present Series		
Right Lung						
Upper lobe	6.7	5.5	6.	5.		
Middle lobe	2.5	3.5	1.	1.5		
Lower lobe	29.8	36.	33.	26.	Cardiophrenic	14.
					Supradiaphragmatic	5.2
					Diaphragmatic	3.4
					Costophrenic	3.4
						26.0
Entire right lung			1.			
Hilus alone	9.1			2.3		
Left Lung						
Upper lobe	5.6	5.	2.5	2.6		
Lower lobe	33.7	50.	43.	41.6	Cardiophrenic	24.
					Diaphragmatic	8.4
					Supradiaphragmatic	7.7
					Costophrenic	1.5
						41.6
Entire left lung			2.			
Hilus alone	3.5			3.9		
Both lungs (lower lobes)	6.0		6.5	12.6	Cardiophrenic	6.6
					Diaphragmatic	3.2
					Supradiaphragmatic	2.5
						12.3
Hilus alone				.3		
Other combinations	3.2		5.	4.5		

Dry cough was often quite persistent, lasting several weeks. Codeine occasionally was necessary, but its use was limited, since it was felt that moderate cough was beneficial physiologically. This cough, in many cases, responded to bed rest alone. In patients showing pronounced bronchial spasm, ephedrine sulfate was beneficial, and in cases with marked tenaciousness of the sputum, potassium iodide was worthy of trial. At times, pleural pain was of such severity as to warrant the use of codeine or even morphine.

The other mode of attack was through the medium of sulfonamide administration. The writers are fully cognizant of the almost universally reported poor results obtained with sulfonamides in this disease.[5, 7, 11, 16] The

drug was not given routinely as Thompson [14] did, but only in those cases exhibiting any one of the following five conditions:

1. Marked febrile reaction of at least 72 hours' duration.
2. Leukocytosis.
3. Clinical pulmonary findings and roentgenogram suggestive of lobar distribution.
4. Failure to show appreciable response to symptomatic management.
5. Suspicion of secondary bacterial invasion, suggested by identification of type specific pneumococci from sputum.

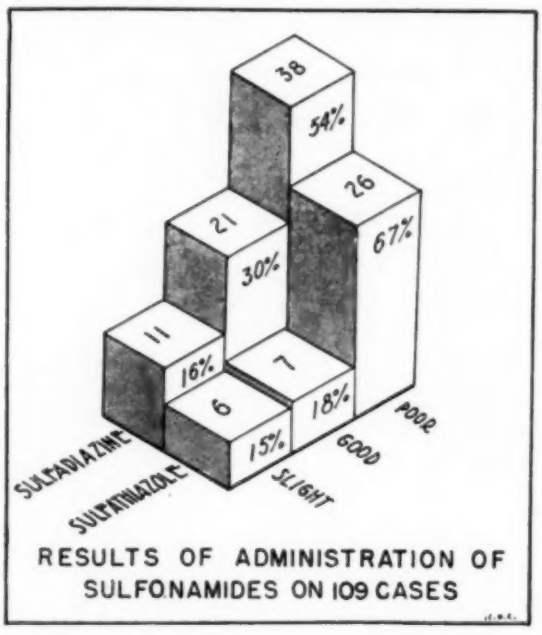

CHART 13.

Our results are depicted in chart 13. Seventy cases were treated with sulfadiazine in the usual dosage. In 38 cases (54 per cent), the result was regarded as poor. In the other 32 cases, 11 showed slight response to the drug, and in 21 cases (30 per cent), the results varied from good to excellent. The results from sulfathiazole were not so satisfactory. Of 39 patients treated with this drug, poor results were obtained in 26 cases (67 per cent), slight improvement in six cases (15 per cent), and good or excellent results in seven cases (18 per cent). In other words, in 109 cases treated with sulfonamides, a good to excellent result was obtained in 28 cases (25 per cent). This is at variance with reports of many other observers.[5, 7, 11, 16] Although atypical pneumonia per se may not often respond to chemotherapy, it is fair to conclude that the disease is frequently complicated by secondary

bacterial invaders against which sulfonamides exert a specific effect. In most cases, the acute stage did not exceed 10 days. As seen in chart 14, the majority were afebrile within five days. In a small number of cases, a temperature of 99.2° to 100° F. persisted for several days to several weeks, occasionally for several months.

The second or subsiding stage was characterized by the absence of fever for 72 hours, the lessening of most symptoms, evidence of clinical clearing of the chest, and roentgenographic changes suggesting resolution. However, the management of this stage occasionally required symptomatic treatment. The dry cough, so troublesome in the acute stage, persisted in few cases and again, simple expectorants, rather than codeine, were indicated. As a further mechanism in the therapy of persistent cough, mild-to-moderate controlled exercise was utilized, and it was felt that this procedure might have been instrumental in reducing the incidence of bronchiectasis. Augmenting pulmonary aeration was particularly indicated, because of the

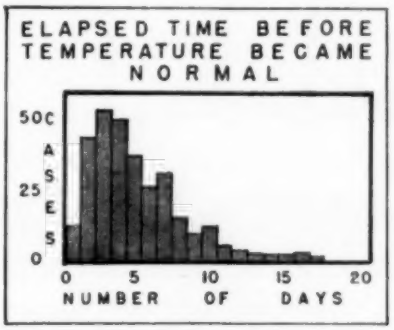

CHART 14.

pathology of this disease. The duration of the subsiding stage was usually between 10 and 21 days.

The final or convalescent stage was reached when the roentgenogram showed complete or nearly complete resolution, the chest findings were negligible, and the patient was asymptomatic, except for varying degrees of weakness and decreased exercise tolerance. Chart 15 indicates respectively the number of days required for the roentgenogram to become clear, the number of days of normal temperature prior to discharge, and the duration of hospitalization. As a rule, the roentgenograms returned to normal much more slowly than did the temperature and physical findings.

As mentioned before, the average time required for roentgen clearance was 18 days. The peaks shown in chart 15 are the result of routine roentgenograms at weekly intervals over a period of four weeks.

The second portion of the chart indicates that on the average the patient was hospitalized for 17 and one-half days after the subsidence of fever. If it is recalled from chart 14 that in most cases the temperature became normal

within five days, the average patient would require a hospital stay slightly in excess of three weeks.

From the third portion of the chart, it is apparent that most patients were hospitalized between 20 and 30 days, averaging 27.5 days. Further hospitalization of roughly 10 days was necessary following roentgenographic clearance because of increased sedimentation rates and delayed convalescence.

Although the convalescent period averaged between 10 days and two weeks, some patients required three to four weeks, and a few needed as much as six weeks or more before recovery was complete. In other words, certain cases presented distinct differences in their ability to combat this disease. The treatment in this stage was graduated exercises and occupational therapy.

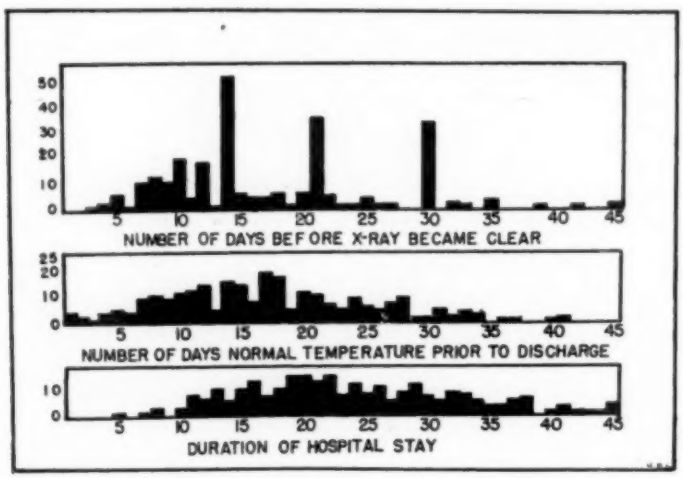

CHART 15.

Repeated attacks of coryza and mild nasopharyngitis were not infrequent, and some of our patients seemed to manifest an increased susceptibility to upper respiratory infections. Particularly was this true in cases which had delayed resolution. Paranasal sinusitis, though not considered a complication, occurred in several cases, but the pneumonic process was not a contraindication to treatment.

A few cases in the subsiding or convalescent stage developed vague abdominal distress. Rarely, this pain became localized in the right lower quadrant. In about eight of these cases, surgical consultation was necessary, and appendectomy was performed in two. This syndrome, often quite persistent, appeared to be independent of the pneumonic process and usually cleared spontaneously.

The following criteria were used in arriving at the optimum time for discharge:

1. Continued normal temperature and absence of pertinent symptoms.
2. Normal chest findings.
3. Roentgenogram within essentially normal limits.
4. Normal sedimentation rate and blood picture.
5. Satisfactory exercise tolerance.
6. Freedom from complications.

By an essentially normal chest film was meant the absence of frank infiltration. The presence of residual prominent bronchial markings, small areas of pleural thickening or diaphragmatic adhesions, were not considered of clinical significance.

Incubation Period and Contagiousness. No conclusion could be reached as to the incubation period of this disease. Various other writers [6, 16] have speculated as to the length of this period, but in our opinion, it is still undetermined.

It is interesting to note that only three members of the medical staff and one nurse contracted the disease during the period included in this study. Despite repeated exposure, no ward attendants employed on the pulmonary service developed the disease.

Complications

In our series, atypical pneumonia was attended by few complications. Of the 321 cases, only 24 (7 per cent) developed a condition which could be so classified.

Symptomatic pleurisy, which is exceedingly common in lobar pneumonia, was rarely observed as a complication. Only seven cases (2 per cent) could be classified as developing frank clinical evidence of pleurisy as a complication. Obviously, many cases had radiographic evidence of pleurisy, manifested by cloudiness of the costophrenic angle or irregularity of the diaphragm, but this was considered an integral part of the picture of the acute disease process and, therefore, not to be regarded as a complication. The type of pleurisy noted roentgenographically was simply a thickening of the pleura as a result of the antecedent pneumonic process. No gross effusion or empyema occurred. This infrequent incidence of wet and dry pleurisy was unusually small in comparison with the incidence reported by other investigators, notably van Ravenswaay,[6] who noted an incidence of 9.7 per cent in pleural effusions alone, and Owen,[8] who observed this complication in 4 per cent. One of Owen's cases developed empyema.

Complications arising from the associated upper respiratory infection were also rare. Only three cases of otitis media, two cases of sinusitis, two cases of conjunctivitis, and one case of laryngitis were observed. Bronchiectasis was also an extremely rare result of the pathological process. This condition was proved to be an actual complication in only two cases. Lipiodol injections were performed in four other suspicious cases, but the bronchograms were considered negative. Blades and Dugan [18] have re-

ported the occurrence of a condition resulting from atypical pneumonia, which they termed pseudobronchiectasis. These investigators considered the lesion to be temporary in most of these cases, returning to normal in a period of four to six weeks. It is possible that the two cases we reported may have been of this type, but in the period of observation permitted, the diagnosis of permanent changes in the terminal bronchial tree was made.

Other uncommon complications were hematuria in one case, persistent headaches of undetermined etiology in two cases, and a recurrence or recrudescence of primary atypical pneumonia in three cases.

Electrocardiographic Abnormalities

Cardiac complications in lobar pneumonia are well-known. Spühler,[19] in routine investigations of the cardiovascular system in lobar pneumonia, found a considerable percentage with cardiac changes. His studies indicated that many of these alterations were mild, reversible myocardial changes evidenced by electrocardiographic and roentgenographic findings. In contrast to these milder cases, irreparable damage, particularly to the conduction system, was observed in other cases. In Spühler's experience, pericarditis was a fairly frequent complication, originating, in his opinion, by extension or metastatic spread.

Early in the present series, these observations were substantiated in a case of lobar pneumonia involving the left lower lobe. During the acute stage of this case, gallop rhythm and a transitory to-and-fro pericardial friction rub were heard. Pericarditis and myocarditis were suggested by the electrocardiogram, manifested by elevated RST segments in all leads, a negative T-wave in the chest lead, and a partial auriculoventricular block with PR interval of 0.32 second. Auriculoventricular conduction time returned to normal, but the elevated RST segments and negative T_{4f} persisted during a three-month observation period. During the latter period of hospitalization, the patient was able to tolerate mild activity. Serial electrocardiographic tracings on this case are shown in figure 3.

Similarly, electrocardiographic abnormalities were observed in one of our earliest cases of atypical pneumonia. These findings served as a stimulus to further investigation of electrocardiographic changes in this disease process. A short time prior to our observations, Fuller and Quinlan [20] and Wolff [21] reported similar changes associated with cases of atypical pneumonia. In 100 cases studied by Fuller and Quinlan,[20] abnormal changes in the RST segments were observed in 11 cases and an inverted T_{4f} was noted in five cases. In their series all of these variations reverted to normal.

Wolff [21] noted almost identical abnormalities in the RST segment in three cases of atypical pneumonia and considered this evidence pathognomonic of pericarditis, despite the absence of clinical findings. Three additional cases of pericarditis associated with primary atypical pneumonia were recently described by Finkelstein and Klainer.[22] The changes in the RST segments in their cases were essentially the same as those noted by the other observers,

and closely resembled the electrocardiographic findings in our cases. In their experience the abnormalities of the RST segment disappeared within a period of three months.

In the aforementioned case (No. 2) typical roentgenographic evidence of a left cardiophrenic type of involvement was present, with only minimal clinical evidence of pneumonia. However, the pneumonic process ran a rather chronic course with persistent low-grade fever of six months' duration. The roentgenogram cleared in a period of about two months. During the fifth month of his illness, the patient experienced vague precordial pains unassociated with dyspnea, cardiac dilatation, murmurs or pericardial friction. Serial tracings were made and figure 4a shows the electrocardiographic

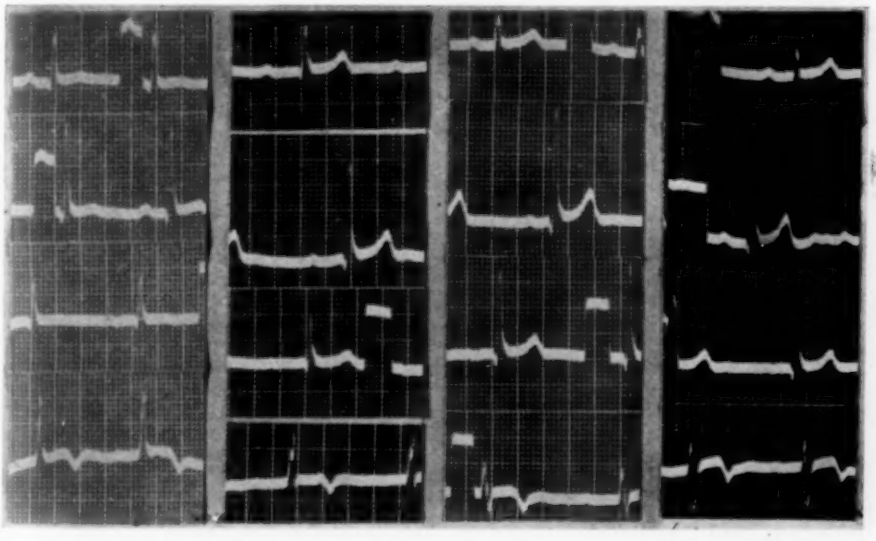

FIG. 3. Electrocardiogram in a case of lobar pneumonia showing abnormalities suggesting pericardial and myocardial involvement.

changes noted in this case. These findings suggested myocardial or pericardial involvement, or both, as evidenced by a negative T_{4f} and a prolonged PR interval. The changes in this case were reversible, and the electrocardiogram returned to normal in a period of about six weeks. Simultaneously, the patient showed clinical improvement and was returned to duty.

Following this initial case, electrocardiograms were done on 63 other cases of atypical pneumonia. Forty cases were studied routinely, utilizing serial tracings, whereas in the remainder, electrocardiograms were done because of either a persistent low-grade fever, unduly delayed roentgen clearance or a prolonged convalescence. Twelve of the cases studied showed electrocardiographic evidence suggestive of myocardial and pericardial involvement. Only two of these patients presented clinical evidence suggesting a cardiac abnormality.

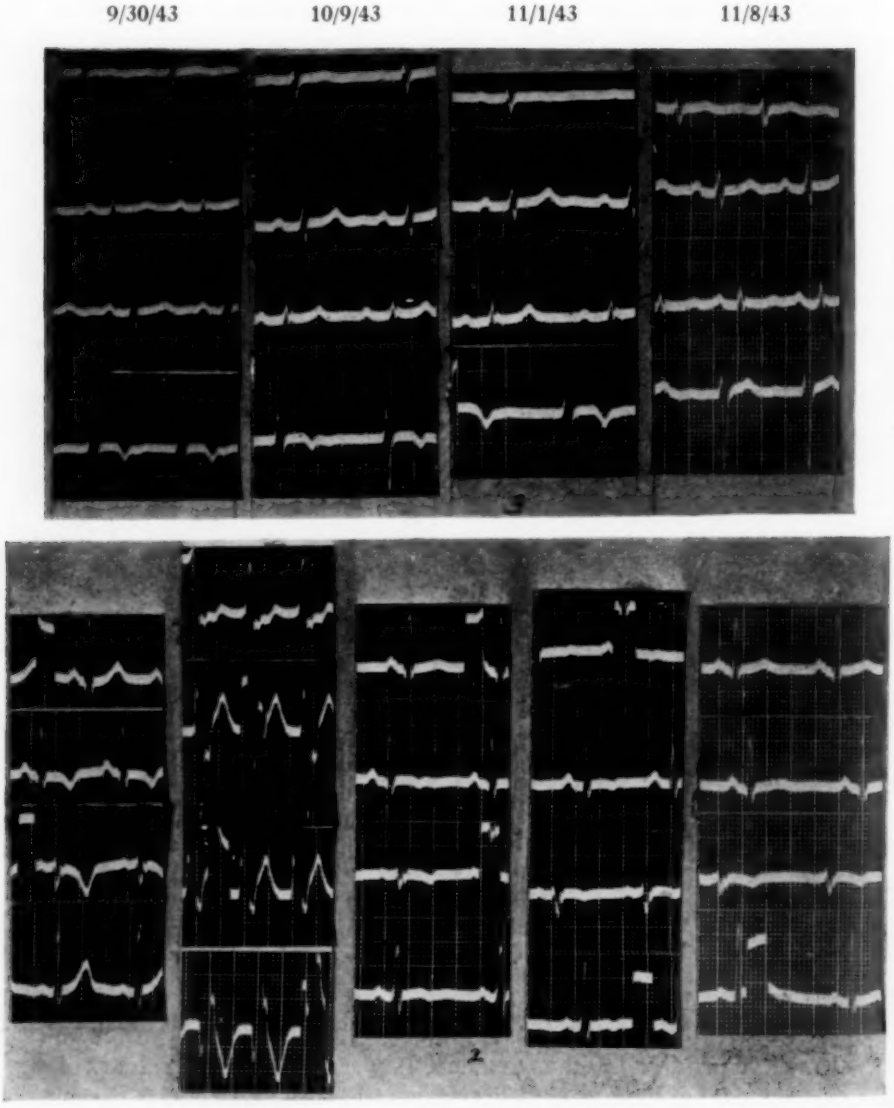

FIG. 4. a (above) and b (below).

Brief case histories and serial electrocardiograms illustrate the typical findings in three other cases (figure 4b, figure 5, a and b).

Case 3, an obese male of 22, was admitted to the hospital with a history of sore throat, fever, malaise, and a dry cough of three days' duration. Physical examination showed only a few subcrepitant râles at the right base, and the roentgenogram revealed minimal infiltration at the right cardiophrenic angle. The latter cleared in two weeks, but a routine electrocardio-

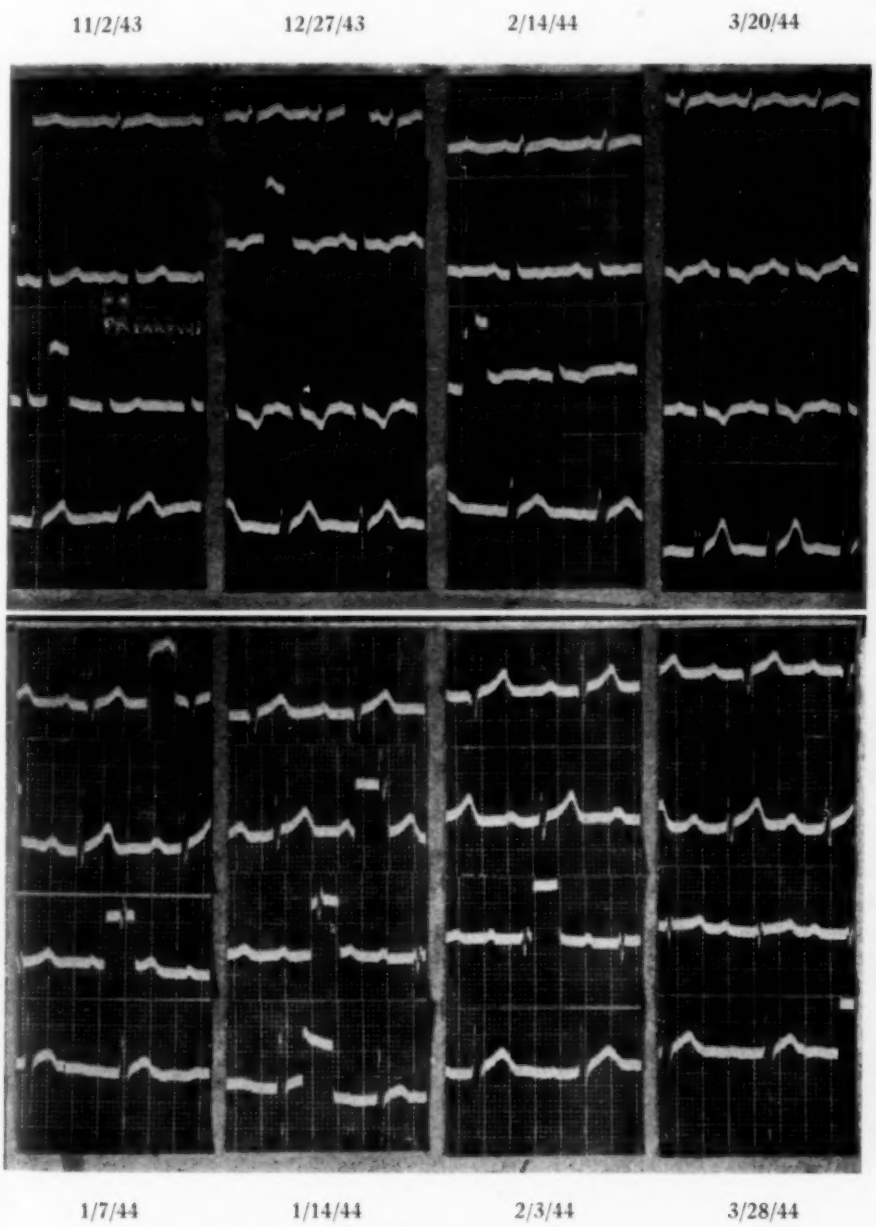

FIG. 5. a (above) and b (below).

gram suggested an active process in the pericardium and myocardium, which reverted to normal only after a period of four months.

Figure 4b (case 3) shows inverted T-waves in the second and third leads in the first set of tracings, nodal tachycardia and bundle branch block in the second, negative T_2, T_3 and T_{4f} in the third, inverted T-waves in all

leads in the fourth, and a reversal towards the normal in the last set of tracings.

The next, case 4, was first observed as an out-patient in the medical clinic, complaining of a non-productive cough of two weeks' duration associated with weakness, cardiac palpitation, and dyspnea on exertion. Clinically, the lungs were clear and the heart negative, except for a moderate tachycardia (100 per minute). Moderate cyanosis was present. An electrocardiogram at this time was normal. A chest film showed a heart of normal size, along with a minimal mottling of the right supradiaphragmatic region. The patient was admitted to the hospital, and the pneumonic process cleared both clinically and radiographically in three weeks, under symptomatic management. The electrocardiograms, however, as depicted in figure 5a, showed changes in T_2 and T_3 which persisted throughout a four month period of observation. During the latter three months in the hospital this patient was ambulatory, afebrile, showed a normal sedimentation rate and except for tachycardia and slight exertional dyspnea, presented no other clincial evidence of cardiac involvement.

The next, case 5, a colored male of 24, was admitted with symptoms suggesting atypical pneumonia, confirmed by roentgenographic evidence in both cardiophrenic angles. This patient was not acutely ill, but exhibited a low-grade fever of 45 days' duration. Roentgen findings required 60 days for clearance. Except for a slight transient precordial pain, there were no symptoms referable to the heart. Cardiac examination was entirely negative. In view of persistent low-grade fever and slow roentgen clearance, along with the complaint of precordial pain, an electrocardiogram was taken.

Figure 5b illustrates the RST elevations observed in this and similar cases. However, in addition to these changes, this patient presented a persistent partial A–V block (PR interval .32 second). Repeated tracings taken over a period of three and one-half months showed no tendency to revert to the normal.

The remaining eight cases showed similar electrocardiographic changes consisting of elevation of the RST segments, inversion of the T-waves, or a disturbance in A–V conduction.

In summation, 12 (3.7 per cent) of the 321 cases studied revealed electrocardiographic deviations from the normal, which consisted essentially of elevation of the RST segments, flattening or inversion of the T-waves in one or more leads, or changes in auriculoventricular or intraventricular conduction. Seven of these cases showed clinical and electrocardiographic reversal to normal, whereas the other five cases showed irreversible changes which persisted throughout a three month observation period. It is important to note that all cases showing electrocardiographic abnormalities were associated with atypical pneumonia limited to the lower lung fields. Seven of these cases occurred with atypical processes involving the left lower lung field, whereas the other five cases developed in connection with disease of the lower right lung. Most of these cases showed atypical processes of

the cardiophrenic type of distribution. It is possible that the proximity of the lesion to the heart may have had a causal relationship to these electrocardiographic changes.

Discussion

For many years any deviations from normal in the RST segments were regarded as unequivocal evidence of organic heart disease. In recent years it has been definitely established that a wide variety of causes, other than organic heart disease, may produce changes in this terminal segment of the tracing. Some of these conditions which have been shown to produce changes in the RST segment or abnormalities in the T-wave are: neurocirculatory asthenia [23, 24]; hyperventilation with alkalosis [25]; effects of various drugs, as digitalis, atropine, mecholyl, prostigmine, epinephrine, nicotine, ergotamine and the sulfonamides; metabolic disorders, as hypothyroidism [26] and hypoglycemia [27]; and blood dyscrasias, i.e. pernicious anemia and acute blood loss.[28] In addition to these causes, various types of infection are capable of altering the RST segment, e.g., rheumatic fever, streptococcal and pneumococcal disease, diphtheria, mumps,[29] virus diseases produced experimentally,[30] trichiniasis,[31] and spirochetosis icterohemorrhagica (Weil's disease).[32]

Lastly, any discussion would be incomplete which failed to include the various forms of organic heart disease that typically affect this portion of the tracing, namely, myocardial ischemia (coronary disease), acute and chronic constrictive pericarditis, acute cor pulmonale,[33] and dissecting aneurysm. A few other less significant conditions could be mentioned but it was felt that they were of insufficient importance to warrant special consideration.

All of the above conditions were considered in determining the significance of these various RST changes. Though neurocirculatory asthenia was a possible clinical diagnosis in some of these cases, the changes in the RST segments were not merely confined to T_2, as reported by Graybiel and White,[23] Merritt [24] and others, but existed usually in several leads. Furthermore, the changes reported in our cases occurred when the tracings were taken in a recumbent posture, whereas those changes reported in neurocirculatory asthenia were noted only when the tracing was taken in a sitting position. Any possible effects of hyperventilation, with resulting alkalosis, can quickly be eliminated, as very few of our patients showed rapid respiratory rates (chart 8). The effect of drugs upon the electrocardiogram can similarly be discounted, as none of our patients received any drugs, with the exception of sulfonamides, that might have an effect on the electrocardiogram. The influence of sulfonamides upon the electrocardiogram deserves a word of mention. It has been shown by Simon [34] that this drug is capable of producing an interstitial myocarditis with localized necrosis in patients dying of sulfonamide intoxication. Inasmuch as only four of our cases in this group received the drug and these cases failed to show any

other evidence of sulfonamide toxicity, the changes reported could not be explained on this basis.

Metabolic disturbances too were considered but pertinent investigation failed to reveal diagnostic evidence of that type of disorder. Blood dyscrasias were not a factor in our series (chart 9).

As mentioned above, changes in the RST segments commonly occur with many infections. Casual observation might lead one to consider these cases actually rheumatic fever with an associated pneumonitis and concomitant myocarditis and pericarditis. It was felt that this possibility could be adequately excluded for the following reasons. First, none of the reported cases fulfilled even the minimal criteria for the diagnosis of rheumatic fever as set up by Jones.[35] It is true our cases presented electrocardiographic evidence suggesting myocarditis but all lacked the supporting clinical findings (significant cardiac murmurs, pericardial friction rubs, etc.) necessary for inclusion under the principal major manifestation. The other major manifestations, i.e., arthralgia and periarthritis, were conspicuously absent. Furthermore, of the minor manifestations only precordial pain was observed and that symptom rarely. Second, none of these cases developed signs of congestive failure, a finding frequently noted in connection with rheumatic pneumonitis. Third, none of our patients at the time these changes appeared developed a significant anemia, leukocytosis, or an appreciably elevated sedimentation rate, commonly seen with rheumatic fever. Fourth, none of our cases in this group gave a previous history of rheumatic fever. Lastly, the relatively non-toxic appearance of the patient and the completely benign clinical course essentially precluded this diagnosis.

The possibility that streptococcal or pneumococcal infection was the basis for these electrocardiographic changes was not likely. All of our cases presented a relatively benign clinical picture and at the time the RST changes were noted the leukocyte counts were within normal limits. It was felt that the mild character of the manifestations and the absence of leukocytosis militated against a streptococcal or pneumococcal origin for these findings. Diphtheria, trichiniasis and spirochetosis icterohemorrhagica could be quickly dismissed as possibilities, because none of the patients showed any of the characteristic findings of these diseases.

Changes in the RST segments, as a result of virus infections, would have to be considered more seriously as a diagnostic possibility. Pearce and Levine[30] have experimentally produced the pathological picture of myocarditis in rabbits by the intratesticular injection of various viruses. These investigators performed electrocardiograms during the early and acute stages of the disease and observed changes in the RST segments with or without disturbances in conduction in 88 per cent of those animals showing evidence of myocarditis at autopsy. Recently Wendkos and Noll[29] reported changes in the RST segment and a prolonged PR interval in a single case of mumps. These observers considered these findings almost unquestionable evidence of myocarditis, despite the lack of symptoms or physical signs.

They pointed out the similarity of electrocardiographic findings in this case with those commonly seen in acute rheumatic myocarditis and considered that a more widespread use of the electrocardiograph is indicated, to determine the true incidence of this complication. It is quite possible that the electrocardiographic changes noted in our series could have been of virus origin but, as previously stated, facilities to prove such a hypothesis were unavailable.

From the electrocardiographic standpoint, several of the tracings could easily be confused with changes resulting from myocardial ischemia. A few of the patients presented electrocardiographic patterns suggesting combined anterior and posterior infarction but the absence of the typical Q-wave pattern and the relatively benign clinical appearance made this diagnosis untenable.

The other conditions mentioned, as acute and chronic constrictive pericarditis, acute cor pulmonale and dissecting aneurysm, can also be quickly excluded as potential causes of these RST changes, as our patients lacked any of the typical roentgen or clinical findings characterizing these conditions.

In conclusion, it is apparent that a small, but definite, number of patients in this series of atypical pneumonia developed distinct changes in the RST segments or T-waves. A few showed disturbances in either AV or intraventricular conduction. Some of these changes were temporary, existing for a period of several weeks or months before reversal to normal. A few of the cases manifested electrocardiographic changes which were irreversible for a period in excess of four months. Strictly from the electrocardiographic standpoint, these changes were highly suggestive of pericarditis, but several investigators [36, 37] have shown that when pericarditis occurs, usually a diffuse subepicardial myocarditis is a concomitant finding. Some of our cases developed evidence suggesting this associated disease, such as both disturbances in conduction and inversion of the T-waves.

Obviously, the etiology of these changes was just as obscure as the etiology of the atypical pneumonic process itself. Inasmuch as none of these cases came to autopsy the full significance of these changes can not be stated with certainty.

Conclusions

1. Three hundred and 21 cases of atypical pneumonia were evaluated in detail.

2. The disease occurred throughout the year, but reached its greatest incidence in the winter months.

3. The onset was usually gradual, non-productive cough and fever being the outstanding symptoms.

4. Crackling râles over the involved area and some degree of pharyngitis were present in roughly two-thirds of the cases.

5. Inspiratory wheezes over the affected area furnished an important diagnostic sign early in the disease.

6. The pulse and respiratory rates were only moderately elevated in most of the cases.

7. A normal blood picture was the usual finding, but leukocytosis, either absolute or relative, did not preclude the diagnosis. Leukopenia and lymphocytosis were equally rare.

8. The sedimentation rate in most of the cases was moderately elevated and was a useful adjunct in determining the time of discharge.

9. A more accurate method of designating the site of involvement was developed. Most atypical processes originated in the hilus and extended into the dependent portion of the lungs. In nearly half of the series, the cardiophrenic angles were the chief site of involvement. The left lung was more frequently involved.

10. Treatment was chiefly symptomatic, but sulfonamides were indicated in selected cases.

11. Complications were infrequent.

12. A small number of cases (3.7 per cent) revealed changes in the RST segments, T-waves or disturbances in conduction, suggestive of pericarditis and myocarditis. Most of these changes were reversible.

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THE MANAGEMENT OF CHRONIC ARTHRITIS AND OTHER RHEUMATIC DISEASES AMONG SOLDIERS OF THE UNITED STATES ARMY *

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DURING the First World War about 93,000 American soldiers developed some sort of "rheumatism." [1] Four common rheumatic diseases—rheumatoid arthritis, rheumatic fever, osteoarthritis and muscular rheumatism—accounted for about 80 per cent of these cases (table 1).

TABLE I

Incidence of Certain Diseases * in the United States Army
First World War (Apr. 1, 1917 to Dec. 31, 1919)
Total of Mean Annual Strengths for the War Period = 4,128,479 Soldiers

Condition	Total Cases	Percentage	Rates Per 1000 Soldiers
Arthritis (rheumatoid and osteoarthritis)	33,613	36%	8.14
Acute Articular Rheumatism † (rheumatic fever)	24,770	27%	6.00
"Muscular Rheumatism"	12,093	13%	2.93
Gonorrheal Arthritis	7,895	9%	1.91
"Myositis"	4,135	4%	1.00
"Synovitis"	3,665	4%	.87
"Tenosynovitis"	2,671	3%	.65
"Ankylosis of Joints"	1,907	2%	.46
"Other Diseases of Joints" (non-traumatic)	1,614	2%	.39
Tuberculous Arthritis	188		.05
Gouty Arthritis	82		.02
Total	92,633	100%	22.43

* These conditions were not fully defined in the original reference; the terms are those used in the Manual of the International List of Causes of Death, Second Revision, Paris, 1909; Washington, D. C., Government Printing Office, 1913.

† In addition to the 24,770 cases of acute articular rheumatism (rheumatic fever), there were 17,372 cases of "valvular heart disease," the great majority of which were probably rheumatic in origin.

The subsequent cost to the government of these 92,633 cases of rheumatism has never been estimated; it must have been very great. In 1931, 13 years after the war, the Veteran's Administration was paying over $10,000,000 a year in disability compensations to about 35,000 ex-Service men with "arthritis" [2] and in 1943, 25 years after the war, the Veteran's

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Administration was expending annually about $2,500,000 in pensions alone to soldiers of World War I with rheumatic heart disease.[3]

Need for Rheumatism Centers

During the First World War no special Rheumatism Center was officially established by the War Department. However, Major Ralph Pemberton and his associates were afforded the opportunity of studying carefully at United States General Hospital Number 9, Lakewood, New Jersey, 400 cases of chronic arthritis among soldiers.[4] "Although General Hospital Number 9 was never designated as a 'Center,' it functioned as one." [5]

During World War I the mean strength of the American Army over a period of two and three quarter years (33 months: April 1, 1917 to December 31, 1919) was 4,128,479. During every month of that period an average of 2807 soldiers developed rheumatism. If the incidence rate for "rheumatic diseases" among soldiers during that war (22.4 cases per 1000 soldiers) were maintained during this second world conflict, the War Department could expect that out of an army of 8,000,000 soldiers, there would develop within the first two and three quarter years of this war (namely, between December 1941 and August 1944, inclusive) about 180,000 cases of rheumatism, an average of about 5454 cases during every month of the war. Of these 180,000 cases, about 64,000 cases would be of chronic rheumatoid arthritis or of osteoarthritis. (Not yet available are the figures for this war.)

To prepare for such a possibility, in the fall of 1942 the Surgeon General and his Associates, with the coöperation of the American Rheumatism Association, took the first tentative steps toward the subsequent establishment of one or more Rheumatism Centers for the army if or when the need materialized.[6] To date the need has been such that five Rheumatism Centers have been established—two for patients with chronic rheumatic diseases and three for patients with rheumatic fever.*

Purposes of a Center

The majority of soldiers who develop rheumatism need not be transferred to special Centers. Patients with transient muscular rheumatism, mild rheumatic fever without carditis, or acute traumatic or specific infectious arthritis can be handled effectively in the adjacent Station or Regional Hospital. Rheumatism Centers are designed for the care of difficult or progressive cases or for diagnostic problems.[7]

The chief aims of a Rheumatism Center are these:

1. Accurate diagnosis: To provide a diagnostic center where difficult cases can be studied by special methods and by medical officers with a special knowledge of rheumatic diseases.

* Three Rheumatism Centers were recently established for the joint Services (Navy, Army, Air Force) of the Canadian Government: one at St. Thomas, Ontario opened in June 1945; others at Winnipeg and at Nanaimo, B. C., opened in April 1945.

2. Intensive treatment: To provide special facilities for the treatment of the more severe or progressive cases.

3. Prompt disposition: To accomplish as great a reduction in hospitalization-time as is consistent with adequate treatment.

4. Increased salvage: To restore to duty, if possible, more men with "cured" or "arrested disease."

5. Rehabilitation: To educate and rehabilitate for civilian life those whose disability necessitates discharge from the army.

6. Application of newer advances in treatment.

7. Appropriate clinical studies of patients while under treatment.

8. Long-range economy, an incidental, but important aim: To reduce the costly need for disability pensions and prolonged hospitalization in Veteran's Facilities.

The Center at the Army and Navy General Hospital

On December 17, 1943 the Surgeon General designated the Army and Navy General Hospital as the first Center for the diagnosis and treatment of rheumatic diseases.[8] This hospital, the Army's oldest general hospital, was chosen because of its past history and excellent facilities. Because of the adjacent hot springs this hospital has, since 1887, been a mecca for the rheumatic personnel of the army. In 1933 the old main hospital building was demolished and replaced by a large new building, and in 1943 an adjacent large hotel was acquired, renovated and connected to the main building, creating a capacity of 1342 hospital beds with an additional 383 beds for patients being reconditioned (total 1725 beds) (figure 1).

A proper knowledge of rheumatic diseases demands familiarity with all phases of general medicine. The productive record of American and European hospitals which have been devoted exclusively to the study of rheumatic diseases has often been disappointing. Rheumatism clinics and services in civilian hospitals maintain their vitality by, and derive much of their inspiration from, their close association with the other clinical and laboratory departments. Therefore, one of the chief advantages of this Rheumatism Center is its placement in a large general hospital with its varied medical and surgical specialties. Thus the rheumatic patient commands the services of specialists in many fields.

The rapid growth of the Center is shown by the daily census of the Section on Rheumatic Diseases which increased from 56 patients present on a given day in January 1944 to 704 patients actually present on a given day in October 1944. During the year 1944, 3105 "rheumatic patients" were admitted, and between January and June 1945 inclusive, 2210 additional "rheumatic patients" were admitted, a total of 5315 in 18 months.

Many of the patients have been received from various camps throughout the country, but during recent months most of the patients have come from overseas hospitals. The majority have come by boat from the South Pacific

or from the European Theater of operations, but many have come via the ambulance planes of the Air Transport Command (figure 2). The speed of evacuation of certain rheumatic patients from overseas has often been startling: some have arrived here by plane within four to six days after leaving South Pacific hospitals (e.g., within four days from Saipan to Hot Springs); others have arrived within three to seven days from England, Italy or France (e.g., from Paris to Hot Springs in three days). Such promptness in evacuating rheumatic soldiers from overseas to hospitals

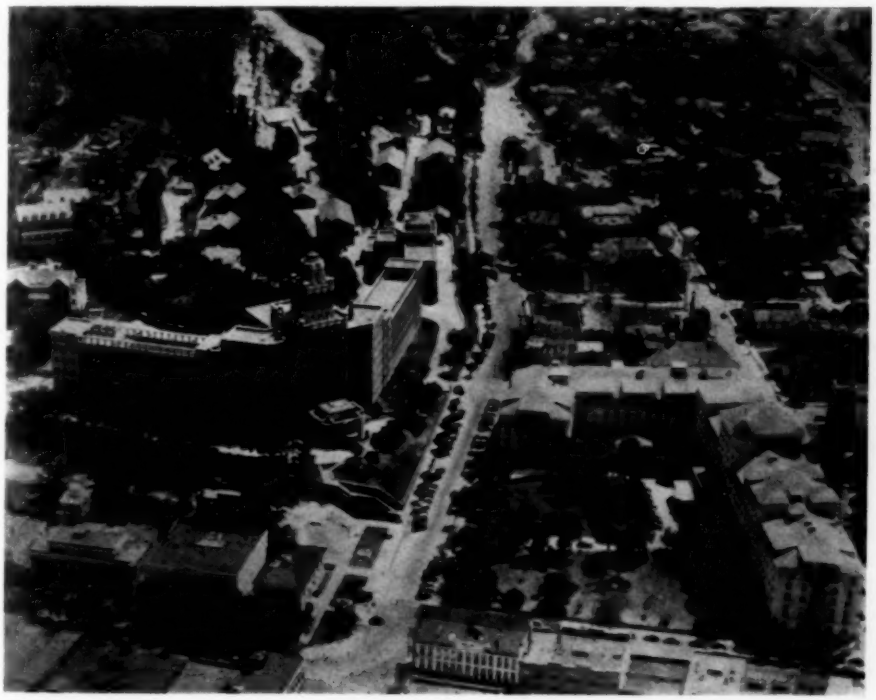

FIG. 1. The Rheumatism Center of the United States Army at the Army and Navy General Hospital; the main building (left) is now connected with the Eastman Annex (right).

equipped especially for their needs fosters a fine morale among the soldiers and their anxious relatives. A proportionate promptness in the subsequent diagnosis and disposition (consistent with adequate treatment) has done much to maintain that morale.

THE CENTER AT ASHBURN GENERAL HOSPITAL

Because the flow of rheumatic patients to the first Center became excessive, a second Center for chronic rheumatic diseases was established August 25, 1944 at Ashburn General Hospital, McKinney, Texas.[9] During its first

eight months the Section on Rheumatic Diseases at that hospital admitted about 2200 patients.[10]

Centers for Patients with Rheumatic Fever

Also on August 25, 1944, the Surgeon General established three Centers for the care of soldiers with rheumatic fever[9]: at Birmingham General Hospital, Van Nuys, California; at Foster General Hospital, Jackson, Mississippi; at Torney General Hospital, Palm Springs, California. Within the

Fig. 2. Arthritic soldiers arriving at The Rheumatism Center after evacuation from overseas in the hospital planes of the Air Transport Command.

first eight months a total of about 900 patients with rheumatic fever was admitted to these three hospitals,[11] most of the patients having been transported thereto by air as soon as possible after the acute phase of the disease began to subside. These 900 patients with rheumatic fever comprised only a minority of the cases of rheumatic fever in the army; during 1942, 1943 and 1944 there were respectively about 1300, 7000 and 6000 cases of rheumatic fever which developed among soldiers in the United States and were recorded by the Surgeon General's office.[12]

The policies of the War Department regarding the diagnosis and management of rheumatic fever have been outlined.[13] The oral administration of

salicylates was considered generally preferable to the intravenous administration. Sulfonamide chemoprophylaxis for the prevention of recurrences was approved; the drug of choice being sulfadiazine, 0.5 to 1.0 gm. daily during the period of convalescence and reconditioning, but not of course during the acute phase of the disease.

The policies of the War Department regarding disposition of such cases are flexible, not static. In general the following patients have been separated rather promptly from Service: those with prolonged active rheumatic fever, those with frequent recurrences and those with significant cardiac involvement. But some patients in the last category who possessed unusual technical skills or other military qualifications have been retained on limited service if their cardiac lesions were well compensated. Most of the 900 patients admitted to these three Centers have been or will be returned to temporary limited duty for six months in a warm dry climate. Thereafter reëxamination and final disposition will be made. To prevent psychic invalidism the War Department has advised that such temporary duty should not involve *undue* limitation of physical activity.

These three Rheumatic Fever Centers belong to the Army Service Forces. In addition, the Army Air Forces has initiated a program of rheumatic fever control in several of their southern Regional Hospitals.[14, 15]

Relative Incidence of Rheumatic Diseases

Because the patients sent to the Rheumatism Center at the Army and Navy General Hospital are selected, our census does not reflect the relative incidence of the rheumatic diseases in the Army as a whole. An analysis of our first 1000 cases has revealed a relative incidence as given in table 2.

Table II

Incidence of Various Types of Rheumatic Diseases among the First 1000 Consecutive Admissions to the Rheumatism Center, Army and Navy General Hospital

	Cases	Percentage
Rheumatoid Arthritis (including rheumatoid spondylitis)	331	33.1%
"Psychogenic Rheumatism"	200	20.0%
Osteoarthritis (primary and posttraumatic)	136	13.6%
Fibrositis (intramuscular and/or periarticular; bursitis, supraspinatus tendinitis, etc.)	134	13.4%
Rheumatic Fever	22	2.2%
Gonorrheal Arthritis	13	1.3%
Gout	10	1.0%
Miscellaneous Conditions (Listed in the order of relative frequency): Sciatica, backache due to ruptured intervertebral disks and other causes, internal derangements of knees, nonspecific monarthritis, traumatic arthritis and synovitis, tuberculous arthritis, psoriatic arthritis, palindromic rheumatism, joint tumors and rare forms of joint disease	113	11.3%
Unclassified Diseases of Joints and Related Structures	41	4.1%
	1000	100 %

A more detailed survey is being prepared for a later report. These figures may be compared to the incidence of rheumatic diseases as seen in a General Hospital which is not a Rheumatism Center.[16, 17]

Here, as in all rheumatism clinics, rheumatoid arthritis presented the main problem; it affected one third of all patients admitted. About one fifth of the patients admitted as "rheumatic" had no significant organic skeletal disease. They suffered from psychoneurosis manifested by musculoskeletal symptoms, a condition called by some "psychogenic rheumatism," [17] by others "psychoneurotic rheumatism" [18] or "psychosomatic rheumatism." [19] This condition will be discussed later herein. Because of the relative youth of soldiers, the incidence of gout and gouty arthritis has been low, the relative incidence being 1 per cent as compared to a relative incidence of 4 or 5 per cent frequently seen in civilian rheumatism clinics. Thanks to modern chemotherapy the total and relative incidence of gonorrheal arthritis has been low.

About one third of our cases of rheumatoid arthritis have been rheumatoid spondylitis, a relative incidence surprisingly high and in notable contrast with experiences in civilian practice. At this Center, for many months we have had at any given time from 70 to 100 cases of rheumatoid spondylitis. The relative frequency of such cases among soldiers probably arises from three factors: (1) Rheumatoid spondylitis affects males much oftener than females, and especially affects young males of military age (18 to 30 years). (2) The early symptoms of the disease, such as vague intermittent low back pain, are difficult to evaluate and an early diagnosis is often not made. Many such early cases in young men have not been recognized until after their induction into the Army. (3) The strenuous physical exertions of Army life and training soon aggravate the symptoms and bring to light these early, previously undiagnosed, cases.

The figures on the relative incidence of rheumatic diseases as seen at our Center are, with a few exceptions, in close agreement with those from the Center at Ashburn General Hospital. Through the courtesy of the Commanding Officer and Staff of that hospital we are permitted to report the relative incidence of "rheumatic diseases" among their first 800 completed cases [10] (table 3).

TABLE III

Incidence of "Rheumatic Diseases" at The Rheumatism Center

Ashburn General Hospital
First 800 Completed Cases

Rheumatoid Arthritis	38.3 per cent
Osteoarthritis	26.7 per cent
"Psychogenic Rheumatism"	16.1 per cent
Postural Backache	2.6 per cent
Internal Derangement	1.8 per cent
Extra-articular Disease (e.g., bursitis) exclusive of fibrositis	1.6 per cent
Specific Infectious Arthritis exclusive of tuberculous arthritis	1.4 per cent
Protruded Intervertebral Disk	1.3 per cent
Tumors	0.7 per cent
Fibrositis	0.6 per cent
Gouty Arthritis	0.6 per cent
Tuberculous Arthritis	0.2 per cent
Miscellaneous	8.1 per cent
	100 per cent

Thus at the two Centers the relative incidences were: rheumatoid arthritis 33 and 38 per cent, "psychogenic rheumatism" 20 and 16 per cent, specific infectious arthritis (including gonorrheal and tuberculous arthritis) 1.9 and 1.6 per cent, and gouty arthritis 1.0 and 0.6 per cent.

Problem of Differential Diagnosis

So relatively inadequate was the general knowledge of the arthritides 25 years ago that a diagnosis of "acute arthritis" or of "chronic arthritis," made without further qualification, was then excusable. Thus the articular and muscular conditions encountered in the First World War were often not clearly defined (table 1). Today an unqualified diagnosis of "acute arthritis" or of "chronic arthritis" is considered inadequate except in rare instances. Although there are many types of acute and chronic arthritis, the rheumatologist and the interested internist should be able usually to subdivide them and state *what kind* of "chronic arthritis" is present. This is a matter of considerable importance in treatment, but especially is it important for a proper estimate regarding prognosis and military disposition.

An analysis of the transfer diagnoses, those with which the patients arrived at the Center, has revealed scores of patients sent here for "arthritis" who had no arthritis at all. Many patients presumably with "osteoarthritis" actually had rheumatoid arthritis, and vice versa. Few of the cases of gout had been correctly diagnosed. A great many of the patients with "muscular rheumatism" actually had, not myositis or fibrositis, but "psychogenic rheumatism"—psychoneurosis manifested by musculoskeletal complaints. Such errors in diagnosis are no particular reflection on medical officers. They merely reflect the diagnostic level of the medical profession as a whole in matters rheumatologic, and exhibit once more the need of physicians in general for a wider and more critical knowledge of fundamentals in the diagnosis of diseases of joints.

Differentiation of Gonorrheal Arthritis from Rheumatoid Arthritis Precipitated or Aggravated by Gonorrhea

It is not sufficiently understood that rheumatoid arthritis can be precipitated by a gonorrheal infection just as it can be precipitated by tonsillitis, influenza or some other acute infection. Also a mild, intermittent or quiescent rheumatoid arthritis can be aggravated by acute genital gonorrhea. Such cases have sometimes been called "post-gonorrheal rheumatoid arthritis" but this condition does *not* represent chronic rheumatoid arthritis engrafted on, or evolving from, a subsiding acute gonorrheal arthritis; it represents simply rheumatoid arthritis precipitated or aggravated by acute genital (not articular) gonorrhea. This entity is not new to the experienced rheumatologist. It is regularly encountered in civilian practice, and in 1 per cent of Pemberton's cases of chronic arthritis among soldiers in the last

World War the arthritis began in close relationship with the onset of gonorrhea.[4]

Proved gonorrheal arthritis among American soldiers in this war appears to be rather rare. In this Rheumatism Center we have seen many more cases of rheumatoid arthritis precipitated or aggravated by gonorrhea than of gonorrheal arthritis. Most of the former cases have been erroneously labelled gonorrheal arthritis, treated as such unsuccessfully by sulfonamides or penicillin or by fever therapy, and transferred to our Center labelled "gonorrheal arthritis resistant to penicillin and/or sulfonamides." In our experience, most cases of so-called "gonorrheal arthritis resistant to chemotherapy" have turned out to be cases of rheumatoid arthritis as shown by their subsequent course, therapeutic tests and, in some cases, articular biopsies.[20] This matter will be the subject of a later report.

Psychoneurosis Manifested by Musculoskeletal Symptoms: "Psychogenic Rheumatism"

Physicians in general are familiar with psychoneurosis as it may affect the gastrointestinal tract (functional dyspepsia, neurasthenia gastrica, irritable colon, anorexia nervosa, etc.) or the cardiovascular system (cardiac neurosis, soldier's heart, neurocirculatory asthenia). Physicians are not so familiar with psychoneurosis as it affects the locomotor system.

"Psychogenic rheumatism," the musculoskeletal expression of functional disorders, tension states or psychoneurosis, is one of the commonest causes of generalized or localized aches and pains in muscles or joints or both, either in civilian or military life. It may exist alone or may occur as a functional overlay of some rheumatic disease such as fibrositis or rheumatoid arthritis. The designation "psychoneurosis manifested by musculoskeletal complaints" is more proper than the terms "psychogenic rheumatism" or "psychosomatic rheumatism." But the term "psychogenic rheumatism" persists in token of its compactness and handiness; if its limitations are understood, it can be a useful label.

Many inadequate, unadaptive soldiers unconsciously make "flight into illness" via musculoskeletal complaints which they, and some physicians also, erroneously call "rheumatism" or "arthritis." These patients have many symptoms but no objective, constitutional, roentgenographic or biochemical manifestations of disease. Actually these patients have no real "rheumatism," no true synovitis, arthritis or organic muscular lesion, or if some minor musculoskeletal disease does coexist it is insufficient to account for the severity of the disability. The clinical pattern is not that of organic rheumatic disease. Typical functional complaints referable to other systems often may be elicited. The degree of psychoneurosis present may vary from a mild anxiety tension state to a major conversion hysteria; camptocormia (hysterical bent back), bizarre gaits, peculiar articular postures or hysterical flexed fingers are not uncommon.

It will be noted that about 20 per cent of our patients and about 16 per cent of those admitted to the Center at Ashburn General Hospital had no significant organic rheumatic disease, at least by the time they reached the Centers (tables 2 and 3). They either had "psychogenic rheumatism" alone, having had no organic "rheumatic disease" at all, or they had a dominating "psychogenic rheumatism" which completely overshadowed an initial and still underlying mild fibrositis or arthritis, or which had completely replaced a previous rheumatic disease (e.g., fibrositis or rheumatic fever), no longer active.

The prompt recognition of "psychogenic rheumatism" is of great importance in the Army to prevent the continuation of the disorder to the point of irreversibility, to prevent unnecessary and unjustified discharge of men on life-time pensions for non-existent "arthritis" or "fibrositis," and, above all, in order to institute the proper methods for the physical and psychic rehabilitation of these unfortunate and generally misunderstood patients.

Differentiation of "Psychogenic Rheumatism" from Fibrositis

Primary fibrositis is the chief rheumatic disease from which "psychogenic rheumatism" must be differentiated. In general, primary fibrositis puts its victims at the mercy of changes in *external* environment: thus weather, heat, cold, humidity, rest, exercise, etc., characteristically influence most of them for better or worse. On the other hand "psychogenic rheumatism" generally puts its victims at the mercy of changes in *internal* environment: thus their symptoms may vary with mood or psyche, pleasure, excitement, mental distraction, worry or fatigue.

Table IV

Tabular Differentiation between Fibrositis and "Psychogenic Rheumatism"; Generalities

	Fibrositis, primary type	"Psychogenic Rheumatism"
General attitude	Coöperative, earnest, "objective"	Tense, anxious, "subjective," defensive, antagonistic
Chief complaint	"Joints hurt and feel stiff"	"Can't quite describe it, doctor. It's like . . ."
Chief symptoms	Aching, soreness, stiffness, fatigue	Burning, tightness, weakness, numbness, tingling, queer or tired sensations
Time of day when symptoms are worse	Morning and/or late afternoon	Inconstant—often continuous day and night
Aggravation or amelioration dependent on:	External or physical environment	Internal or mental environment
Effect of mental preoccupation: (theatre, movie, bridge, etc.)	No definite relief, symptoms intrude	Often marked relief but perhaps "pays for it afterwards"

TABLE IV.—*Continued*

	Fibrositis, primary type	"Psychogenic Rheumatism"
Symptom Analysis		
1. Pain:		
Amount	+ to ++	+ to +++
Constancy	Varies in intensity during day: worse in morning, better at noon, often worse again later in day	Tendency to be constant, "bad all the time"
Duration	Hours or days Remissions, exacerbations	Momentary or constant, "no different," getting worse.
Location	Anatomical	Often not anatomical
Migration	May not migrate; if so migrates in anatomical fashion	Bizarre, hemalgia, etc.; may follow no anatomic pattern
2. Stiffness	Worse after much rest (jelling). More marked in early morning. Better after mild exercise.	Minimal or not present. Jelling not characteristic.
3. Fatigue	A.M. on waking: 0 to + P.M. ++ "Disability causes fatigue"	Early A.M. + to +++ May be constant "Fatigue causes disability"
Effect of rest	After prolonged rest—worse (jelling)	Improvement or no effect
Effect of exercise	Better "limbers up"	Worse during and after
Effect of applied heat	Temporary relief—hours	Variable—often worse
Effect of weather	Worse when cold and damp. "Weather prophet."	Variable
Effect of therapy:		
In general	Temporary relief	"Nothing helps me, doctor"
Patient's attitude	Admits relief	Defies finding a cure
Aspirin	Temporary relief—hours	Usually no relief (aspirin futility), or "never tried it" (aspirin inutility)
Physical therapy	Temporary relief	Variable—often worse
Response to examination:	Coöperative; tenderness consistent	Fearful, resistant; "touch me not" reaction
"Extras" (associated functional complaints)	0 to +	+ to ++++ Bizarre limps and postures, headaches, globus hystericus, sighing respirations, precordial pains, insomnia, nervousness, tremor, etc.

Space does not permit the inclusion here of more than a tabular differentiation in general terms (table 4). The differentiation depends, of course, not on any one feature but on a combination of features. When a case of one or the other disorder is relatively "pure," differentiation is readily made. Differentiation and a correct assay of the problem are especially difficult when a mild fibrositis coexists with a marked functional overlay. Nevertheless this differentiation has been very useful to us.

Treatment

The comprehensive schemes of treatment, used at this Center, for the various rheumatic diseases are those approved by the American Rheumatism Association [21] and used by the leading rheumatologists of the country. This Center does have unusual facilities for physical therapy and hydrotherapy; these facilities are used properly but without undue emphasis and certainly not to the exclusion of any other useful measure.

Group lectures: Rheumatic victims are, in general, docile, patient and well-behaved. Of their physicians they ask surprisingly little; in lieu of the elusive "rapid-cure," they ask only for a diagnosis and a decent understanding of what they are up against, what they can do to help themselves, and what they should not do lest they make themselves worse. They will abandon the physician who brushes them off with an incomplete diagnosis or a fancy diagnosis in medical terms and "a few well chosen words." To answer their need, we have instituted here a regular rotating series of group consultations or "lectures on rheumatism" given in laymen's language. Of the 12 different lectures, two or three are on general topics for all patients; others are given to the appropriate groups of (generally 25 to 100) patients with a particular disease. One group of patients is usually not admitted to the specific lectures designed for another group. Especially are patients with "psychogenic rheumatism" *not* permitted to attend the lectures for patients with rheumatoid arthritis or fibrositis lest misinterpretations arise. Instead, those with "psychogenic rheumatism" hear special talks designed for their particular needs and given jointly by a rheumatologist and psychiatrist.

The lectures are on the following subjects: (1) The meaning of rheumatism and arthritis; (2) facts, fads and false concepts about rheumatism; (3) fibrositis—its meaning and management; (4) rheumatoid arthritis and its management; (5) rheumatoid spondylitis and its management; (6) facts about osteoarthritis; (7) gout and gouty arthritis; (8) shoulder disabilities and their management; (9) body mechanics in relation to disability of joints; (10) home physical therapy (motion picture and demonstration); (11) emotional tension and its relation to "rheumatism"; (12) the management of rheumatic fever.

These group consultations are not a substitute for, but supplemental to, individualized consultations. They are designed to project beyond the period of Army hospitalization and into the patient's home at least some of the benefits he may derive from the more formal treatments here. They also serve as an introduction to the advice which each patient will later receive from his home physician. The lectures have been well received, and incidentally, have been a great time-saver for the busy medical officer. After each lecture the patients are encouraged to ask questions on points that bother them, no matter how trivial they may seem; any question about something not understood is a valid question. The lectures also improve morale:

seeing that he is not alone in his problem and that others are worse than he, the patient takes courage.

Rheumatoid arthritis: Our treatment for this disease is quite standard and includes the removal of obviously infected foci, the use of highly nutritious diets (but there is no "anti-rheumatism vitamin" or specific diet), foreign protein therapy in selected cases, simple analgesics, physical therapy, occupational therapy, orthopedic measures to prevent or correct deformities, gold salts carefully administered to selected patients whose rheumatoid arthritis is progressive in spite of more conservative measures, and roentgen therapy for certain cases of rheumatoid spondylitis. We found penicillin to be ineffective.[22]

Psychogenic rheumatism: The treatment of psychogenic rheumatism has been an interesting but difficult problem, second here in importance only to that of the treatment of rheumatoid arthritis. Our pleasure at being able to reassure soldiers with psychogenic rheumatism that they do not have arthritis or muscular rheumatism and that they need not fear the presence of a crippling disease is tempered by the difficulty of helping them to develop insight and to accept their diagnosis, at least to the point of submitting whole-heartedly to a trial of psychotherapeutic reconditioning. In these cases the latter is of much greater value than physical reconditioning; physical reconditioning used alone in these cases accomplishes little or nothing.

Patients with psychogenic rheumatism are not generally given formal courses of physical therapy or other treatments used for "organic rheumatism" except as diagnostic or therapeutic tests, because such treatments often tend to fix more firmly in their consciousness the belief that they have organic disease.

The Disposition of Rheumatic Soldiers

Nothing could destroy a soldier's potentialities for salvage (his morale, his will to recover and to serve) more readily than the atmosphere of a "chronic hospital," a "rheumatic old soldier's home." The rheumatic soldier should not be kept in a state of prolonged uncertainty as to whether he will probably be returned to duty (limited duty, if necessary) or whether he will be discharged from the Army. Whatever his future is to be, it should be, relatively speaking, an immediate future, not a vague distant future. No hasty dispositions should be made, but in most cases it does not take long to determine the probable disposition required for a given rheumatic disease or the future military potentialities of a given rheumatic soldier. Furthermore, "right or wrong," a disposition should be made fairly promptly, unless prolonged definitive treatment is indicated; otherwise the rheumatic soldier may develop the hospital habit, the outlook of the dependent chronic invalid or pensioner. If hospitalized too long, a salvable patient with a mild form of rheumatism may, even though he was originally well oriented, develop some form of hospital-engendered psychoneurosis or

fixation of illness, and the functional overlay may become more difficult to treat than the original organic disease upon which it became superimposed.

To combat these possibilities, each patient on arrival is told that his stay is decidedly not indefinite, that his period of hospitalization will follow a rather definite and progressive, though elastic, schedule: a few days for a thorough initial physical survey, then a period of intensive treatment (generally about three to eight weeks, longer in selected cases), after which it will be decided whether he can be "reconditioned" for further military service or should be "rehabilitated" for civilian life.

Reconditioning: Getting a convalescent soldier physically and mentally prepared to return to military duty is spoken of as "reconditioning." [23] The salvable rheumatic soldier is "reconditioned," first in the hospital by the medical program noted above, then by a supplemental period of two or more weeks during which time he lives in a convalescent barracks and undergoes daily a program of physical activity carefully measured to his abilities. Some patients recovering from a transient rheumatic disease can participate in a fairly strenuous program; for other patients who at best can only be expected to return to limited service, the reconditioning program is less strenuous; in every instance an attempt is made to apply the program as an individual prescription.

Rehabilitation: If a soldier's rheumatism precludes the possibility of his return to duty within a reasonable period of time, if his disease is essentially progressive and disabling, the soldier will be made ready for discharge to civilian life and for subsequent follow-up treatments by his civilian physician or, if necessary, by a Veteran's Facility. To such a soldier, as to the one who can return to duty, the Army acknowledges an equal obligation: getting him prepared mentally and physically to return to a useful civilian life, despite his rheumatic disability, is spoken of as "rehabilitation."

After his discharge from the Army, the arthritic patient may have to modify the patterns of his life somewhat, so as to avoid factors known to be aggravating to his disease, but he must not alter his life to the point of engendering defeatism. The educational program mentioned heretofore is one of our chief weapons against the dangers of a wheel chair—or crutch-psychology. The discharged arthritic patient must still regard himself as a vital unit of his community. We therefore attempt to teach him how to live with his disease, not for it. As long as possible, the treatment of his rheumatism should be merely an avocation, not his vocation. If prematurely or needlessly he makes a vocation of his disease, he has taken a long step toward the sterile existence of the pensioner's rocking chair.

Policies: Each of the rheumatic diseases poses its own problem in disposition.

For patients recovering from acute rheumatic fever the disposition must take into account the presence or absence of rheumatic carditis or the likelihood of its early development. The need for manpower has been such that blanket discharges for rheumatic fever could not be entertained. Disposi-

tions have been individualized.[13] The general policy in force at the Rheumatic Fever Centers has been mentioned.

Most patients with rheumatoid arthritis, certainly those with progressive disease, should be discharged. However, we have attempted to salvage the mildly affected patients whenever possible; otherwise we cannot learn to what extent salvage is feasible or to what extent the Army can utilize the soldier with a clear brain and a stout heart, but with slightly rheumatic joints.

The disposition of patients with "psychogenic rheumatism" requires individualized consideration. Many soldiers affected with psychoneurosis of mild or moderate degree can still render effective service. But we are ordered to conserve, not men, but manpower. When, despite conscientious treatment, "psychogenic rheumatism" persists to the extent that its victim no longer represents a unit of manpower, then he is recommended for discharge, because a man without power is a drag on the Army.

Statistics on Disposition

For this report we have summarized the dispositions made on 1300 cases, not chosen serially but selected only so as to include a representative number of cases of each of the commoner rheumatic diseases (table 5). Of

Table V

Disposition of 1300 Soldiers with Rheumatic Disease
The Rheumatism Center, Army and Navy General Hospital

Condition	Patients	Returned to Duty (full or limited duty)		Separated from Service by Medical Discharge or Retirement	
		Patients	Per cent	Patients	Per cent
Rheumatoid Arthritis, including Rheumatoid Spondylitis	500	76	15.2	424	84.8
"Psychogenic Rheumatism"	200	128	64.0	72	36.0
Fibrositis, primary	150	123	82.0	27	18.0
Osteoarthritis	100	38	38.0	62	62.0
Rheumatic Fever	50	39	78.0	11	22.0
Gonorrheal Arthritis	20	13	65.0	7	35.0
Gout	10	1	10.0	9	90.0
Miscellaneous and Unclassified Cases of Arthritis and "Rheumatism"	270	171	63.4	99	36.6
Total	1300	589 patients = 45.3%		711 patients = 54.7%	

the 1300 patients, 589 or 45.3 per cent were returned to duty of some kind, to full duty or to temporary or permanent limited duty; 711 or 54.7 per cent were discharged (enlisted men) or retired (officers) from service. Thus about half of the patients were returned for a further trial of duty. In contemplating these preliminary results one must keep in mind the fact that military duty, even limited duty, is pretty strenuous business, unsuited for

those who cannot work regularly at least eight hours a day. The concessions that can be made to the rheumatic soldier are limited.

A follow-up study is being made of both groups: of the retained group to note to what extent our attempts at salvage were successful; of the discharged group to note the further course of the disease when the patients were freed from the physical and psychic stresses of Army life.

It will be noted that most of the patients with primary fibrositis, "psychogenic rheumatism," rheumatic fever or gonorrheal arthritis were returned to duty, whereas the majority of those with rheumatoid arthritis, osteoarthritis or gout were separated from service. Many of our osteoarthritic patients were elderly commissioned or non-commissioned officers of long services in the Army. In our opinion most gouty patients are not suitable for Army life, considering the difficulties of following a medicinal and dietary regime and the likelihood of encountering provocative physical trauma. Frequent recurrences of acute gouty arthritis make such persons of limited or doubtful military value.

Clinical Investigation

"It is hoped to make this hospital a source of extensive knowledge on arthritis for the whole medical profession. Studies will be carried on in the use of special drugs, such as sulfonamides and penicillin, in the treatment of arthritis": so read the War Department's announcement of the establishment of the first Rheumatism Center.[7] Thus the Army acknowledged an obligation to the arthritic soldier, not merely as an individual, but as a representative of all his kind. Thus the medical officers serving at the five Rheumatism Centers are encouraged to improve our clinical knowledge of the rheumatic diseases, to improve, if possible, our methods of treatment, and to present clearly the results to the medical profession. In an effort to fulfill this obligation a number of clinical investigations are being carried out at each of the five Centers. Although quite young, the two Centers for chronic rheumatic diseases are already, so far as we know, the largest Rheumatism Centers in the world. As treatment Centers and carefully supervised schools of rheumatology, they are providing a unique opportunity which should benefit mutually both the rheumatic soldier and his medical officer.

Summary

"While arthritis does not account for a large percentage of illnesses in the United States Army, it has been found to be one of the most disabling": so the War Department has stated.[7] During the First World War about 93,000 American soldiers developed some type of "rheumatism," an incidence rate of 22.4 per 1000 soldiers. The incidence of rheumatic diseases among soldiers in the Second World War has been such that the Surgeon General

has established five Rheumatism Centers for selected cases: two for chronic rheumatic diseases, three for rheumatic fever.

The management of rheumatic soldiers at one of the Centers has been outlined. Rheumatoid arthritis, psychoneurosis manifested by musculoskeletal symptoms ("psychogenic rheumatism"), primary fibrositis and osteoarthritis were the conditions most often encountered. The high relative frequency of rheumatoid spondylitis and of "psychogenic rheumatism" were of special interest. Gonorrheal arthritis among soldiers appears to be relatively uncommon. More often seen were cases of rheumatoid arthritis precipitated or aggravated by acute genital (not articular) gonorrhea. It is believed that many, if not most, cases of so-called gonorrheal arthritis resistant to sulfonamides or penicillin or both are in reality cases of rheumatoid arthritis precipitated or aggravated by, or coincident with an otherwise unrelated genital gonorrhea.

Psychoneurosis manifested by musculoskeletal symptoms, "psychogenic rheumatism," has presented a common and difficult problem and affected 15 to 20 per cent of the soldiers, presumably "rheumatic," admitted to the two Centers for chronic rheumatism. The recognition of "psychogenic rheumatism" is of importance in military life in order to initiate effective treatment promptly and to prevent unnecessary discharges and pensions for nonexistent "arthritis" or "muscular rheumatism." Psychogenic rheumatism must be differentiated especially from primary fibrositis (muscular or capsular rheumatism). In general fibrositis puts its victims at the mercy of changes in external environment (weather, heat, cold, humidity, rest, exercise) whereas "psychogenic rheumatism" tends to put its victims at the mercy of changes in internal environment, symptoms being altered for better or worse by changes of mood or psyche, by pleasure, excitement, mental distractions, worry, or fatigue. The clinical differentiation has been outlined briefly.

Of 1300 "rheumatic patients" disposed of at one of the Centers for chronic rheumatism, 45 per cent were returned to some type of military duty; 55 per cent were separated from Service.

The five Rheumatism Centers are providing an unusual opportunity to give the rheumatic soldier the best available study and treatment and to advance the knowledge of rheumatic diseases.

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RHEUMATIC HEART DISEASE IN NEW GUINEA: INCLUDING A CARDIOVASCULAR SURVEY OF 200 NATIVE PAPUANS *

By Harold D. Levine, Lt. Col., A.U.S., F.A.C.P.

In October 1944 Major A. M. Harvey of Baltimore showed the writer a case of rheumatic heart disease with mitral and aortic stenosis and insufficiency in a native with acute cardiac decompensation at the Native Hospital conducted by Angau (Australia-New Guinea Administrative Unit) at Lae, Mandated Territory of New Guinea. This patient died a few weeks later but permission for a postmortem examination was not obtained. In view of the prevalent belief that rheumatic fever and rheumatic heart disease are only exceptionally encountered in the tropics, this case was considered a medical rarity. However, in January 1945, when the author had the privilege of accompanying a Malaria Research Unit of the United States Army in an investigation of natives along the Papuan Gulf, the opportunity was taken to investigate the frequency among them of rheumatic heart disease and any other abnormalities of the cardiovascular system.

For this purpose a group of 200 native Papuans of random age and sex distribution living in the villages of Uritai and Seapiapi was examined. The location of these villages is shown on the map (figure 1). The examination of these individuals included palpation of the brachial, radial, temporal and dorsalis pedis arteries, ophthalmoscopic examination, careful examination of the heart, lungs and abdomen, and determination of the blood pressure with an anaeroid sphygmomanometer. Hemoglobin estimations were made by the Tahlqvist method in all individuals with murmurs. Although the inaccuracy of this method is appreciated it is felt that from it a qualitative statement is warranted that the subject does or does not have anemia. Electrocardiograms and roentgenograms of the chest were not feasible. In view of the fact that the determination of the left border of cardiac dullness by percussion would not be universally acceptable as evidence of heart size, a statement regarding heart size was not made unless the position of the maximum apical impulse of the heart was felt. In view of the lack of evidence of cardiac displacement from physical examination, location of the apex impulse to the left of the midclavicular line was arbitrarily accepted as evidence of cardiac enlargement. Murmurs were graded on the basis of one to six, grade one being the faintest murmur audible on careful auscultation and grade six, one which may be heard with the naked ear at some distance from the chest.[1]

Accurate records were available on the ages of all individuals in the younger age group, but the age given for the older members of the com-

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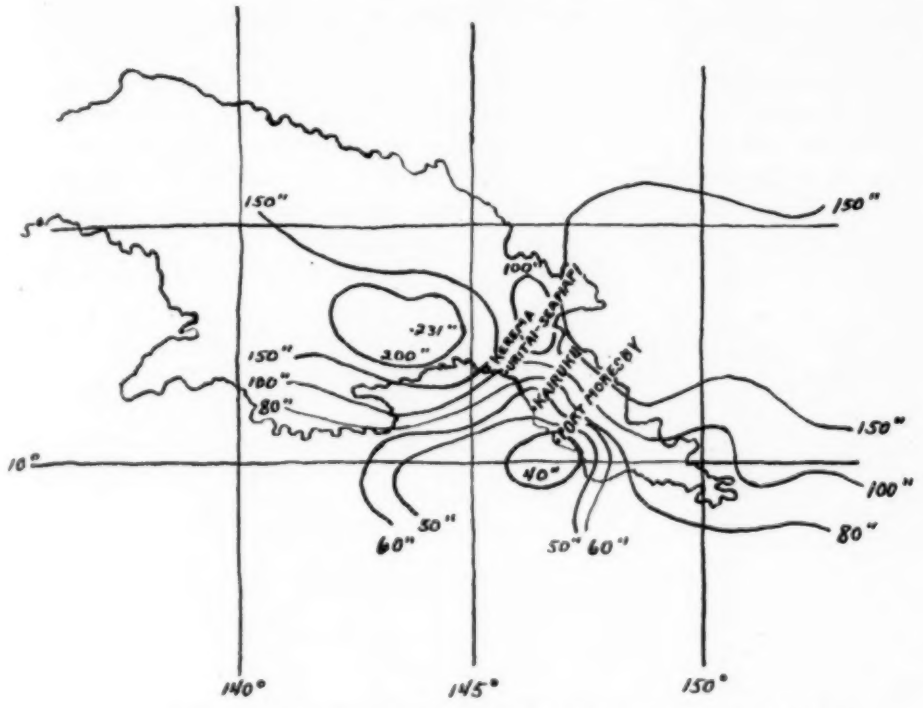

FIG. 1. Isohyetal map of eastern New Guinea showing the location of the villages studied.

munity is only approximately correct. The scarcity of elderly individuals is at once apparent in chart 1. Only 20 people (10 per cent) were more

CHART I

Age Distribution of Individuals with Cardiac Murmurs, Cardiac Enlargement and Peripheral Arteriosclerosis

Age Group	0–5	6–10	11–15	16–20	21–25	26–30	31–35	36–40	41–50	51+	Total
Number of Individuals	23	40	50	15	16	9	9	18	13	7	200
Murmurs Due to Rheumatic Heart Disease	0	2	0	0	0	0	0	0	0	0	2
Murmurs Due to Anemia	4	7	8*	3	4	1	0	1	0	1	29
Unexplained Systolic Murmurs	0	0	3	1†	0	0	0	0	0	0	4
Cardiac Enlargement Due to Anemia	2	0	2	0	0	0	0	0	0	0	4
Unexplained Cardiac Enlargement	0	0	0	0	1	0	0	1‡	1‡	0	3
Peripheral Arteriosclerosis	0	0	1	2	4	3	1	6	7	5	29
Retinal Arteriosclerosis	0	0	0	0	0	0	0	0	0	0	0

* The murmur in one of these individuals was also explicable on the basis of fever, tachycardia and active dermatitis.

†"Possible heart disease": other members of this patient's family also had murmurs.

‡ Each of these individuals had peripheral arteriosclerosis.

than 40 years of age. From a superficial examination one would say that the population apparently ages rapidly. A man or woman of 30 or 35 will usually already have developed some of the stigmata of senility, loss of elasticity of the skin, arcus senilis or thickening, tortuosity or prominence of the visible arteries. Random notes taken at the time of this study state "this man of 30 looks 50," "a wizened old fellow of 35," or "a grizzled matron of 39 with peripheral sclerosis and arcus senilis." There were 108 females and 92 males in the sample studied.

The Detection of Cardiac Murmurs. In 165 individuals no murmurs

CHART II

Auscultatory and Palpatory Findings and Hemoglobin Values in Individuals with Heart Murmurs

Murmur					PMI cm.*	MCL cm.*	HB % (T)	Splenic Index	Interpretation of Murmur	Interpretation of Enlargement
No.	Age	Sex	Grade	Location						
1	10	F	1 systolic mid-diastolic rumble	Apex Apex	—	—	50	3	Mitral stenosis. Rheumatic heart disease	—
2	9	M	2 systolic 2 systolic mid-diastolic rumble	Apex 2nd & 3rd lis Apex	8.5	6.3	70	3	Mitral stenosis and insufficiency. Rheumatic heart disease	Same
3	12	F	3 systolic and thrill 2 systolic	Base trans. to neck Apex	10.0	6.5	50	3	Unexplained. ?Aortic stenosis	Unexplained
4†	14	M	2 systolic 1 systolic	Apex lsm	8.0	7.2	70	0	Unexplained. ?Mitral insufficiency. Migratory polyarthritis at 7	Same
5	16	F	2 systolic 2 systolic	Base Apex	—	—	70	0	"Possible heart disease"	—
6†	13	M	1+ systolic (musical)	Apex	—	—	80	1	Unexplained	—
7	15	M	2+ systolic	Apex	—	—	60	2	Anemia	—
8	7	F	2 systolic (musical)	Apex	—	—	60	3	Anemia	—
9‡	14	F	2 systolic 1+ systolic	Apex 2nd lis	9.0	7.5	60	3	Anemia	Anemia
10	30	F	2 systolic	Apex	8.0	5.8	50	3	Anemia	Anemia
11‡	21	M	2 systolic	Apex	10.0	8.0	70	2	?Anemia	Anemia
12	22	M	2 systolic	Apex	—	—	70	0	?Anemia	—
13	11	F	1 systolic 2 systolic S_1 snapping	Apex 1st & 2nd lis	—	—	55	2	Anemia	—
14	3	M	2 systolic	3rd & 4th lis	—	—	60	2	Anemia	—
15	14	F	2 systolic	2nd lis	—	—	60	2	Anemia	—

* A hyphen in these columns indicates either that the apex impulse was not felt, or, if felt, was not located to the left of the midclavicular line.

† Numbers 4, 6 and 35 are brothers.

‡ Numbers 9, 11, 16 and 40 are siblings. A third sister had a normal heart without murmurs.

lsm = left sternal margin. lis = left interspace.

PMI = Position of maximal cardiac impulse to the left of the midsternal line.

MCL = Distance from midsternal line to midclavicular line.

CHART II—*Continued*

Murmur					PMI cm.*	MCL cm.*	HB % (T)	Splenic Index	Interpretation of Murmur	Interpretation of Enlargement
No.	Age	Sex	Grade	Location						
16‡	16	F	1 systolic 2 systolic	Apex 2nd lis	9.5	7.6	60	2	Anemia	Anemia
17	20	F	1 systolic 1 systolic	Apex Base	—	—	50	2	Anemia	—
18	6	F	1 systolic	Apex	—	—	50	3	Anemia	—
19	24	M	1 systolic	Apex	—	—	50	2	Anemia	—
20	14	M	1 systolic	Apex	—	—	50	2	Anemia	—
21	25	M	1 systolic	Apex	8.5	7.3	60	0	Anemia	Anemia
22	36	M	1 systolic	Apex	8.5	8.0	60	0	Anemia	Anemia
23	5	M	1 systolic	lsm	—	—	50	2	Anemia	Anemia
24	10	M	1 systolic	lsm	—	—	50	2	Anemia	—
25	13	F	1 systolic	lsm	—	—	50	3	Anemia, fever, tachycardia and dermatitis	—
26	17	F	1+ systolic	3rd & 4th lis	—	—	50	3	Anemia	—
27	8	M	1+ systolic (musical)	3rd lis	—	—	50	2	Anemia	—
28	14	F	1 systolic	3rd & 4th lis	—	—	50	4	Anemia	—
29	15	M	1 systolic	3rd lis	8.5	6.3	50	3	Anemia	Anemia
30	5	M	1 systolic	lsm	6.5	5.0	60	3	Anemia	Anemia
31	5	M	1 systolic	lsm	—	—	60	2	Anemia	—
32	6	F	1 systolic	1st & 2nd lis	—	—	60	2	Anemia	—
33	10	F	1 systolic	2nd lis	—	—	70	2	Anemia	—
34	52	M	1 systolic	4th lis	12.5	8.0	70	2	Anemia	Anemia
35	10	M	1 systolic	2nd & 3rd lis	—	—	70	1	?Anemia	—

were heard. Thirty-five individuals had systolic murmurs of some intensity. Of these, two had apical diastolic murmurs as well. The location and intensity of these murmurs is given in chart 2. In this group of 35 individuals 29 had systolic murmurs of grade 1 or 2 intensity associated with hemoglobin values of 70 per cent or less. Malaria was hyperendemic in this area as evidenced by spleen survey and blood smears, and hookworm infestation was found in 24 per cent of all individuals studied in these villages.[2] The operation of at least these two factors made anemia quite a common finding in this group. It is well established that anemia can produce systolic murmurs and cardiac dilatation.[3, 4, 5, 6, 7, 8, 9] Therefore, the murmurs found in this group of 34 individuals having only grade 1 or 2 systolic murmurs were not considered diagnostic of valvular disease but were arbitrarily ascribed to the anemia.

There were four individuals whose systolic murmurs were regarded as "unexplained." The first was a girl of 12 (Case 3) with a harsh grade 3

systolic murmur at the base of the heart maximal at the left of the sternum, a palpable thrill in the same area, and a grade 2 systolic murmur at the apex. The position of the maximum impulse (PMI) was located 3.5 centimeters to the left of the midclavicular line (MCL) in the fourth left intercostal space. The hemoglobin value was 50 per cent and the splenic index 3. Although aortic stenosis was suspected, that diagnosis was not hazarded in view of the anemia. The second was a boy of 14 (Case 4) with slight cardiac enlargement, grade 2 systolic murmur at the apex, an independent systolic murmur along the left sternal margin, a hemoglobin of 70 per cent, and a splenic index of zero. His mother said that he had been incapacitated because of pains and swellings in his joints for several months when he was seven years old. This patient is regarded as one who, on being followed for a number of years, might well eventually develop unequivocal evidence of organic valvular disease. The third was a girl of 16 (Case 5) with grade 2 systolic murmurs at apex and base, a booming first heart sound at the apex, a third heart sound at the apex, no cardiac enlargement, hemoglobin 70 per cent, and splenic index zero. She is the sort of patient who would probably be listed as "possible heart disease" in a Cardiac Clinic. The fourth was a boy of 13 (Case 6) with a grade 1 musical systolic murmur at the apex, no cardiac enlargement, hemoglobin 80 per cent, and splenic index 1.

Two patients in the group had definite evidence of organic rheumatic valvular heart disease. The first, a girl of 10 (Case 1), had a grade 1 systolic murmur and a definite long mid-diastolic rumble at the apex. The apex impulse was in the midclavicular line, the hemoglobin 50 per cent and the splenic index 3. The second was a boy of nine (Case 2) with a grade 2 soft systolic murmur at the apex, another grade 2 systolic murmur in the second and third intercostal spaces just to the left of the sternum and a definite long low-pitched mid-diastolic rumble at the apex culminating in a sharp presystolic crescendo whip and a booming first heart sound. The apex impulse in this case was 2 centimeters to the left of the midclavicular line in the fourth interspace, the hemoglobin 70 per cent, and the splenic index 3. In the absence of evidence that anemia can produce the diastolic rumble characteristic of mitral stenosis, each of these individuals was regarded as having organic mitral stenosis, and, surely the second, mitral insufficiency as well.

At this point it may be interpolated that following the studies at Uritai and Seapiapi a visit was made to Kerema, further along the Papuan Gulf. At the Angau Native Hospital there the writer was shown a case diagnosed as endocarditis by Sgt. James M. McKerrell, E. M. A. (European Medical Assistant). The patient, a man of about 35, had mitral stenosis and insufficiency and aortic insufficiency with auricular fibrillation, apparently of recent origin and the cause of his admission. Including the patient seen at Lae and the two children at Uritai and Seapiapi, this brings to four the number of natives of New Guinea in whom the diagnosis of rheumatic heart disease was made.

Cardiac Enlargement without Heart Murmurs. In nine individuals without heart murmurs the apex impulse was felt to the left of the midclavicular line (chart 3). In six the enlargement was attributed to anemia. In three the enlargement was unexplained. Two individuals in the latter group had peripheral arteriosclerosis.

Blood Pressure Determinations. The systolic blood pressure determined in 137 individuals 11 years of age or older varied from 106 to 162 with a mean of 106 millimeters. The individual with a systolic pressure of 162,

CHART III

Cardiac Enlargement in Nine Individuals without Heart Murmurs

No.	Age	Sex	PMI	MCL	HB %	Splenic Index	Interpretation of Enlargement
36	40	F	9.0	6.5	50	2	Anemia
37	12	M	8.5	6.5	60	3	Anemia
38	5	F	6.5	5.5	60	1	Anemia
39	13	M	8.5	6.0	60	2	Anemia
40*	18	M	11.5	7.5	70	0	?Anemia
41	14	M	8.5	7.0	70	2	?Anemia
42	37	M	9.0	7.5	80	0	Unexplained. Peripheral arteriosclerosis. BP 114/78
43	47	M	8.5	7.5	80	0	Unexplained. Peripheral arteriosclerosis. BP 106/62
44	24	M	11.0	6.8	80	0	Unexplained

PMI = Position of maximal cardiac impulse to the left of the midsternal line.
MCL = Distance from midsternal line to midclavicular line.
* Numbers 9, 11, 16 and 40 were siblings. A third sister had a normal heart without murmurs.

a man of 62, was the only one in the entire group whose systolic pressure exceeded the "normal range." His diastolic pressure was 78 millimeters. In the entire group the diastolic pressure varied from 40 to 90 millimeters with a mean of 65 millimeters. Two individuals had "borderline" diastolic pressures. One was a girl with a blood pressure of 120 mm. Hg systolic and 90 mm. diastolic. The other was a 40 year old man with slightly thickened brachial and radial arteries and a blood pressure of 120 mm. Hg systolic and 90 mm. diastolic. As shown in chart 4 the range of blood pressures and the

CHART IV

The Blood Pressure of 137 Papuan Natives

Age Group	11–25 Years	26 Years and Older	Total
Number of Individuals Examined	81	56	137
Systolic Blood Pressure			
Maximum	132 mm.	162 mm.	162
Minimum	82	85	82
Mean	105	106	106
Diastolic Blood Pressure			
Maximum	85	90	90
Minimum	40	50	40
Mean	65	62	65

mean blood pressures were essentially the same when the group was "broken down" into two age groups: 11 to 25 years of age, and 26 years of age or older. The figures were also practically identical when considered by sex.

There were 38 individuals in the entire group with a systolic pressure of 100 millimeters Hg or less. In this group the systolic pressure ranged from 82 to 100 with a mean of 95 millimeters, and the diastolic between 50 and 76 with a mean of 60 millimeters. In the group of 137 individuals in whom the blood pressure was taken then, 27.7 per cent had hypotension. These figures were closely approximated in both age and sex groups.

Vascular Disease. Some degree of thickening and tortuosity of the brachial, radial, temporal or dorsalis pedis arteries was noted in 29 individuals. In 16 it was slight and in 13 moderate in severity. All but two of these were males. The exceptions were a woman of 39 with moderate and one of 46 with slight peripheral sclerosis. The age distribution of individuals with sclerosis of the peripheral arteries is shown in chart 1.

In contrast to the frequency of peripheral arteriosclerosis were the ophthalmoscopic findings. None of the 56 individuals 26 years of age or older showed retinal vascular changes. Ophthalmoscopic examination was not carried out in the younger age groups.

The Meteorological Background. In view of the alleged relationship between climate and rheumatic fever it was considered of interest to review the meteorological features of this part of the Papuan coast. The writer is indebted to the 15th Weather Squadron, 42nd Weather Station, AAF, for supplying the data given below.

1. Rainfall. There is considerable variation in the amount of rainfall along the Papuan coast. The vicinity of Port Moresby is the driest with an annual average of 40 inches. Proceeding along the coast there is a steady increase in the annual fall reaching a peak at Kikora, at the head of the gulf. Figures are available from the stations at Kairuku on Yule Island, approxi-

CHART V

Monthly Averages of Rainfall (in Inches) over a 20–23 Year Period

	Jan.	Feb.	Mar.	Apr.	May	June	July	Aug.	Sept.	Oct.	Nov.	Dec.	Year
Kairuku	9.99	10.28	8.56	5.22	1.53	2.11	1.03	0.65	1.62	1.54	2.92	5.59	54.04
Kerema	9.44	8.09	10.68	11.26	16.92	16.33	13.12	13.93	12.47	12.32	9.27	7.35	141.18

mately 60 miles, and from Kerema (chart 5), approximately 130 miles northwest of Port Moresby. The villages studied, Uritai, and Seapiapi, lie about halfway between these two stations.

Moving Northwest along the coast to Kerema there is a sharp increase of annual rainfall due to certain topographical conditions. The dry and wet seasons are less pronounced, there being considerable rainfall even in the so-called dry season. As the isohyetal map (figure 1) shows, the annual rainfall in the Uritai-Seapiapi region is about 100 inches.

2. Temperature and Humidity. Along the coast of Papua, daytime maximum temperatures show a definite, though small, monthly variation, being highest in the months of November to March and lowest in June, July and August. The annual range of maximum temperatures averages about 8 degrees (F.) over all coastal stations.

Over the Papuan coastal regions the humidity is uniformly high, rarely falling below 70 per cent. Kerema (chart 6) is the only station in the area under discussion for which any data on temperature and humidity are available. It is obvious from these figures that we are dealing, in the region studied, with a hot humid climate with relatively little variation.

CHART VI

Temperature and Humidity at Kerema

	Yrs. of Record	Jan.	Feb.	Mar.	Apr.	May	June	July	Aug.	Sept.	Oct.	Nov.	Dec.	Year
Mean of Daily Max. Temps. (deg. F.)	11	90.2	90.3	89.6	88.1	86.8	84.8	83.8	83.4	84.8	86.8	88.6	90.1	87.3
Mean of Daily Min. Temps.	11	73.8	73.7	74.2	74.4	74.3	73.7	72.5	72.6	73.3	74.2	74.1	74.1	73.7
Relative Humidity at 9 a.m. (%)	16	79	77	79	80	84	86	86	87	86	84	80	80	82

Dietary Considerations. Living as they do on a diet of sago, a few tropical vegetables and fruits such as cocoanut, breadfruit, pawpaw and limes, no milk, some fish, and meat only on very rare festival occasions, it seems likely that these people suffer from some degree of dietary deficiency of one type or another. Unfortunately, a careful search was not made for clinical evidence of vitamin deficiency, but chance observations were occasionally made of rachitic deformities of the chest and perléche.

COMMENT

Cardiac enlargement and systolic bruits of the type described above have been observed in the anemia of hookworm disease [9] and chronic malaria.[6, 7] In view of the fact that most of the individuals in the surveyed group had malaria and many also had hookworm infestation, it seems likely that most, if not all, of the systolic murmurs detected are attributable to these causes. In one investigation [10] soft blowing diastolic murmurs have been ascribed to severe anemias, but there is no convincing evidence that rumbling mid-diastolic murmurs or presystolic crescendo murmurs are attributable to anemia. It seems tenable then that all four of the individuals described actually had organic mitral stenosis.

The observations presented on the blood pressure and vascular status of these individuals are obviously too limited to warrant general conclusions, but the cases of valvular heart disease, by virtue of their mere existence in any number in this limited series of cases, seem worthy of comment. It is

quite well established that the percentile incidence of rheumatic fever and rheumatic heart disease is smaller in the southerly than in the northerly regions of the United States.[11, 12, 13, 14, 15, 16, 17] These conditions have been regarded as diseases of cold, damp and stormy [16] environments and their inception in the tropics has been regarded as questionable [18] or extremely rare and with mild manifestations and less striking sequelae.[19, 20] In recent years, however, there have appeared more and more case reports of rheumatic fever and rheumatic heart disease in tropical [21, 22, 23, 24, 25] or semitropical [26, 27, 28] regions. The extreme view has even been taken [23, 27] that they are as frequent in the tropics as in the temperate zones. Autopsy statistics on this point are meager. Major T. C. Backhouse [29] in a series of about 1400 necropsies performed at Rabaul from 1925 to 1940 found two cases with fish-mouth mitral valves. Microscopic examination of one of these hearts in Australia showed typical Aschoff bodies. The available literature reveals only one previous report of rheumatic heart disease in New Guinea.[30] This was in a youth of 18 in cardiac failure in whom physical examination suggested mitral stenosis, the origin of which was regarded as a mystery.

Streptococci are regarded as involved in one way or another in the natural history of rheumatic fever, but data on their incidence in the pharyngeal flora of healthy residents of the tropics or those suffering from acute infections of the upper respiratory tract, are contradictory. Beta-hemolytic streptococci were cultured far less frequently in Puerto Rico than in New York City [31] and much less frequently from natives of Rabaul [29] and from white troops in Australian [29] and American [32] general hospitals in the Mandated Territory of New Guinea than in temperate regions, but Norris [33] found beta-hemolytic streptococcus 77 times in 272 cultures from cases of upper respiratory tract infection among the armed forces in the South Pacific. Further clinical, pathological and bacteriological studies along these lines might throw some light on the still highly controversial subject of the etiology of rheumatic fever.[34]

Summary and Conclusions

1. Four cases of mitral stenosis were observed in Papua and the Mandated Territory of New Guinea. Two of these were detected in a cardiovascular survey of 200 native Papuans living in a hot damp environment. The other two, who were patients in native hospitals, also had auricular fibrillation.

2. Rheumatic heart disease is apparently uncommon, but by no means rare, in natives of eastern New Guinea.

3. It is felt that the more carefully people in the tropics are studied, the more universal will rheumatic heart disease be found to be.

4. Systolic murmurs and palpable cardiac enlargement, explicable on the basis of anemia, were frequently detected.

5. Despite the apparent absence of retinal vascular disease and infrequency of hypertension, the population ages prematurely. This is apparently

due to the prevalence of the infectious tropical diseases of youth rather than to the degenerative changes of middle age.

Addendum: Two interesting contributions have appeared in the literature since this article was written. The writers of the first paper [35] in describing 20 cases of rheumatic carditis among 1307 autopsies at Curaçao, Netherlands West Indies, rightly insist that the true incidence of rheumatic carditis can be determined only by collecting reliable data, based especially on autopsies with histological examinations. The authors of the second paper [36] raise the question of an actual increase in the incidence of rheumatic fever in Panama since about 1927.

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THE DOCTOR AS A WITNESS *

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The Different Capacities in Which the Doctor May Be Called to Testify

There are three different sets of circumstances under which a doctor may be called as a witness and the rights, privileges and duties of the doctor will be found to vary with those circumstances. They are as follows:

1. Where the doctor is possessed of information relevant to some issue in the case, which information he acquired in other than a professional capacity. Example, the doctor, while driving to make a call, sees an automobile hit a pedestrian. The pedestrian sues the automobile owner for damages for negligence. The doctor may be called by either party to testify on the issue of negligence.
2. Where the doctor has treated a patient and he is called upon to testify as to the physical condition of the patient, the treatment administered, etc.
3. Where the doctor has never treated the person whose bodily condition is in issue but is called as an expert to assist the court in arriving at the determination of a scientific fact as to the bodily condition of such person, the expectation as to his recovery, etc.

Of course, the doctor may often be called as a witness in more than one of the above capacities. In the example mentioned as capacity No. 1, the doctor who sees the pedestrian hit may stop and treat the injured man. In capacity No. 2, the doctor, in addition to testifying as to his treatment of the patient, may be called upon to advise the court as to his opinion of the present condition of the patient and the expectation of his recovery.

Nevertheless, the above distinctions as to the capacity in which the doctor is to be called upon to testify must be borne in mind in determining certain questions as to his duties, responsibilities and privileges as a witness.

The Duty of the Doctor to Testify

It is the duty of every citizen to testify when called upon by a court of law, regardless of any personal inconvenience to the witness, and this duty may be enforced by a court writ known as a "subpoena," a writing served upon the prospective witness commanding him to appear and testify in a certain court in a certain cause and on a certain named day. Although there is some question whether a doctor may be compelled to testify in capacity No. 3 without extra compensation (this question will be discussed later), as to capacities 1 and 2 the doctor is no different from any other man

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and *must* respond when summoned regardless of other professional engagements.

The manner of service of the subpoena varies in different jurisdictions. It is usually served by a court officer but may be served by any person, including the litigant himself or his attorney. The service must be accompanied by the tender to the witness of the statutory fees, so much for each day's attendance (a small amount, usually $1.50 or so), plus mileage from the place of residence of the witness to the courthouse. The failure of the witness to respond to the summons contained in this writ is punishable as contempt of court, the punishment being either a fine or imprisonment.

Attorneys generally realize that the doctor is a very busy man, who should not be compelled to sit around a court-room, day after day, waiting to be called to the stand, and they will usually be considerate in informing the doctor that he need not appear at the exact time named in the subpoena but that if he will promise not to go out of town without letting the attorney know in advance that he is to be away and will arrange that no matter where he is he can be reached by telephone on short notice, the attorney will arrange to telephone him when he is actually needed. If the subpoena is served by someone other than the attorney, the doctor should telephone the attorney and endeavor to make the arrangement above suggested. If, however, the attorney is unwilling to so arrange, the doctor must be there at the court-room at the time named, regardless of the other calls upon his time.

When the doctor reaches the courtroom and is not called to the stand right away he should get in touch with the attorney at the first recess (there is usually a short recess in a trial in the middle of the morning and in the middle of the afternoon, as well as the recess at noon and that at the end of the day) and ask the attorney to call him to the stand as soon as possible so that he can get back to his patients. Often under such circumstances the attorney will obtain leave from the judge to call the doctor out of turn, sometimes, for such purpose, temporarily withdrawing from the stand another witness whose examination has not yet been concluded.

To reassure the doctor who has never been a witness and who may find himself dreading what he considers as an ordeal, it should be explained that very rarely is a doctor ever called to the stand "cold," without ever having gone over with the attorney the matter of what his testimony is to be. Usually there will have been at least one conference between the two, often more than one, in which conference the doctor will have been advised as to the questions that will be asked him and will have told the attorney what his answers to such questions will be. If the issue is an important one, where the reputation of the doctor may be in any way at stake, the doctor should insist on having such a preliminary conference.

Privilege Not to Testify

As stated above, the doctor has a duty to testify when called upon. Has he a *privilege* not to testify?

The answer to the above question is that the one certain privilege of the doctor is the privilege of any witness not to be compelled to answer a question put to him when the effect of that answer may be to incriminate the witness. This, it will be noted, is not a privilege to refuse to take the stand, but a privilege to refuse to answer a certain question. As to whether an answer to a question might incriminate him is for the witness himself to decide. The method of claiming such privilege is very simple. The witness says to the judge, "I decline to answer such question on the ground that the answer might tend to incriminate me."

There is also, in certain jurisdictions, a privilege of the witness not to answer the question if the effect of the answer will be to disgrace the witness, although not actually to incriminate him. The ruling on such claim of privilege will depend upon whether the judge considers the question relevant to some issue in the case. If it is relevant, the answer can be compelled. If it is not relevant, the claim of privilege will be sustained.

As has been stated, the above are the only privileges of the doctor. What about what is popularly known as the doctor-patient privilege? That is the privilege of the patient only and it in no way belongs to the doctor.

In discussing the doctor-patient privilege it should first be pointed out that such privilege is not a so-called "natural" privilege recognized by the courts from ancient times but is a privilege that exists only because so ordered by a particular legislature. It exists, if at all, only by statute, and its extent is exactly measured by the language of the particular statute. A statute on this subject will be found to have been enacted in something over one-half of the states of the Union and, as these statutes will be found to differ somewhat in their terms, an explanation of the rule on this subject in a given jurisdiction will have to depend upon a study of whether there is a statute on such subject in that jurisdiction and what its language is.

Many of the statutes are, however, fairly similar in their wording and a general idea of such legislation can be gained by examining the statute of New York, which was the first state to legislate on this subject. It reads as follows:

"A person duly authorized to practice physic or surgery, or a professional or registered nurse, shall not be allowed to disclose any information which he acquired in attending a patient in a professional capacity, and which was necessary to enable him to act in that capacity; unless, where the patient is a child under the age of sixteen, the information so acquired indicates that the patient has been the victim or subject of a crime, in which case the physician or nurses may be required to testify fully in relation thereto upon any examination, trial or other proceeding in which the commission of such crime is a subject on inquiry." [1]

Analyzing such statute it will be noted

(1) The statute applies only to persons duly authorized to practice medicine or surgery, or a professional or a registered nurse.[2] The person consulted must be a professional physician in the usual sense of the word.

It does not include a veterinary surgeon or a pharmacist. In view of the modern recognition of dental science as a branch of medical science it should include a dentist, although there is authority to the contrary.[3] It includes a practitioner of any branch or school of medical science recognized as such by the law or the reputable medical profession. Whether it applies to a licensed osteopath or chiropractor is a matter of dispute.[4]

(2) The consultation with such a person must be had in his professional character at the time. A consultation for some purpose other than that of ultimate curative or alleviative treatment is not privileged; nor is a communication made at some time when the professional relation is not pending. A communication made to a medical practitioner invited to an inspection or consultation at the opponent's instance is not privileged, because it is not usually made for the purpose of curative treatment,[5] although if such practitioner does undertake to administer treatment the privilege does attach.[6]

(3) The information acquired must have been necessary to enable the doctor to treat the patient. The word "information" has been broadly construed so as to include data furnished through submission to inspection as well as oral communications.[7] But it is the tenor or substance of the communication only that is privileged. The mere fact of making a communication as well as the date of a consultation and the number of consultations are therefore not privileged from disclosure so long as the subject communicated is not stated.[8]

Statements made to the doctor which were not necessary as the basis for any treatment to be administered are not privileged. For example, a person injured in a street accident who is picked up by an ambulance tells the ambulance surgeon en route to the hospital the story of how the accident occurred. The disclosure is not privileged.[9] A doctor called to give first aid to a person injured in an automobile collision was allowed to testify that he noticed the odor of liquor on the man's breath.[10]

The privilege exists regardless of whether the doctor received any compensation for his services or rendered them with any expectation of receiving compensation.

As previously stated, the privilege is that of the patient, not of the physician. The latter cannot claim the privilege if the patient is willing to waive it. The waiver may be made in a number of ways. It may be expressed or implied. The patient may waive it on the trial. He may waive it in advance of the trial and in advance of any litigation by consenting in an application for a life or accident insurance policy that any conversation had with the physician regarding the physical condition shall not be privileged.[11]

The patient may waive the privilege by his conduct, this involving what the law calls an "implied waiver." As the whole theory of the privilege is based upon the patient's supposed unwillingness that his ailment should be exposed to the world at large, where the patient himself discloses to the world his physical condition the privilege should no longer be recognized. For example, the patient himself calls the doctor to the stand and examines

him regarding his physical condition; or the patient requests the doctor to act as a witness to his will, knowing that the doctor will be called upon to testify as to the patient's physical condition when the will is offered for probate; or the patient calls another doctor to the stand to testify as to the patient's physical condition; or a patient sends in a doctor's certificate as part of his proof of claim for benefits under an accident insurance policy; or his beneficiary sends in such a certificate with his proof of claim on a life insurance policy; or a patient sues a physician for malpractice. In all these cases it can be logically contended that the privilege has been waived, although the courts have not been consistent in so holding.[12]

The privilege between doctor and patient survives death, but it can be waived by the executor, administrator, or an heir of the decedent. It may also be waived by the guardian of a minor or of an insane person.[13]

As heretofore stated, the existence of the privilege and its extent will depend in every case upon the decision of the legislature as to whether it wishes to pass such a statute and how far that statute should go. It will be found, also, that even in a state which has established the privilege, the legislature has almost invariably made it inapplicable to workmen's compensation cases, and modern sanitary legislation has also abolished the privilege, in part, for venereal diseases and for narcotic drugs.[14]

The above explanation as to the law of privilege has been given for the benefit of a doctor reader, but it is not at all necessary that he should attempt to make himself familiar with the rules on this subject or their distinctions. Usually the patient will be a party to the law-suit. If he himself calls the doctor as a witness there is no difficulty, as he declares himself thereby as being willing to have the doctor testify. If the opponent calls the doctor as a witness, the counsel for the patient will himself make the objection unless he is willing that the privilege be waived. The only situation for which the doctor must be on the look out is the one where the patient is not a party to the cause and there is no other party to look after his interests. In that case the doctor will listen to the question addressed to him and if, in his opinion, it calls for a conversation with the patient that might be privileged, he will simply turn to the judge and say, "Your honor, I doubt if I ought to answer that question in view of the fact that it calls for a statement that I consider privileged." The judge will either say, "You need not answer," or he will explain that the testimony called for is, for some reason, not within the privilege rule and he will instruct the witness to answer. If so instructed by the judge, it will be the duty of the witness to answer the question and he can do so without any disturbance of conscience. Whether there is a privilege against disclosure under those particular circumstances is purely a question of law and on that question the person to decide is the judge.

How a Witness Is Examined in a Legal Proceeding

The term "legal proceeding" is used in the above heading advisedly instead of "court," for a doctor will be called as a witness not only before courts

but before coroners in their inquests, before administrative tribunals, such as industrial accident boards, and in statutory proceedings for the commitment or release of the insane. In general, the method of examination of the doctor as a witness will be the same in all proceedings, the only difference being that before administrative tribunals the proceedings are likely to be less formal and the rules of evidence are much less strictly applied than in a court. Also there is often less technicality applied when the hearing is before the judge sitting as the sole trier of fact than when the case is tried before a judge and jury. In the last named case the jury decides all questions of fact. The judge decides all questions of law.

In any form of legal proceeding, the witness is first sworn to tell the truth and is then examined by the party calling him. The examination is by question and answer. The questions are usually short, if calling for some specific fact, but sometimes a question will be put in a broad form, such as, "When you went to the patient's house on the day you mentioned please tell us what happened?" To this question the witness will give what is called a "narrative" form of answer, going ahead with a story of what did happen until he finishes or is interrupted by the judge or by counsel. A familiar form of question put to a doctor which calls for a narrative reply would be, "When you first saw the patient on the day you have stated what did you find his condition to be?"

Opinion Evidence

Facts are called for and not the opinions or conclusions of the witness. The reasons for this rule are obvious. The function of the trier of fact, whether the judge sitting alone or the jury, is to reach the proper conclusion from the facts of the case. Therefore, as a basis for his conclusions what this trier of facts wishes are facts, not opinions. For example, in a suit for damages for negligence arising out of an automobile collision, a witness can never be asked, "Was the defendant negligent?" Negligence is a conclusion, often reachable only on the basis of many facts. The information that the court does wish from the witness is, "What did the defendant do?" "How fast was he driving?" "What was the condition of the traffic light?" "Where was the plaintiff?" "What was he doing?"

There are certain matters, however, on which the trier of facts is not able to reach a conclusion without help from someone, for no matter how many facts may be brought to his attention he will not know how to apply such facts so as to reach a correct conclusion. These are mainly cases where scientific principles are involved and the law not only permits but welcomes help from men versed in that particular science to aid the trier of facts in arriving at a proper conclusion. This help can be given not only in instructing the court as to the principles of the particular science but in giving the court the opinion of the witness as to the proper conclusion from the fact under the rules of that science. If the witness knows more than does

the court about a science whose principles are involved in the issue on trial, he is an "expert witness." An expert need not be filled with book learning. He may have acquired his special knowledge by long experience. Also there are, of course, borderline cases where it is difficult to determine whether the question is one for expert opinion or not, whether by general experience the jury do not know a simple scientific fact anyway without asking for aid. For example, a man loses a finger on a circular saw and the issue is his own carelessness. Is it a scientific fact on which a court will receive opinion testimony that when a saw is whirling the outer edge is invisible to the eye, or is that something which every juryman knows and on which expert testimony is not needed?

Opinions by Medical Experts

On medical questions, however, there is never any doubt as to the necessity of receiving opinion testimony. Any doctor, by reason of his training, is competent to advise the trier of fact as to the intricacies of the medical art and is therefore an expert witness. The doctor need not have had any experience of his own on the particular medical question involved in the case. His education and training alone fit him to act as an expert. Nor does he need to be a specialist in any particular branch of his profession. He does not have to be an orthopedic surgeon to testify as to the proper way to set a broken limb, nor a psychiatrist to testify as to the sanity of a patient. The more experience a doctor witness may have had and the more learned he is in the particular field the better, but this goes only to the weight of his testimony, not to the admissibility of it.

Hearsay Evidence

Another rule of the law of evidence is that what the court wishes to hear is what the witness himself knows, not what he may have heard from others. This is what is called the rule against hearsay. Ordinarily a witness cannot testify, "I know it is true because John Smith told me." The court will say, "We do not want to get this second-hand from you. Produce John Smith and let him tell his story here so that he may be cross-examined upon it."

The hearsay rule is not an absolute one, however, for there are certain matters that may not be susceptible of proof in any other manner than by hearsay. For example, a man cannot ordinarily tell how old he is or who his parents are except by hearsay. Therefore the law has had to create an exception to the hearsay rule, which exception is known as the "pedigree" rule. Statements of deceased members of the family as to family relationships are admissible even though hearsay. There are various other established exceptions to the hearsay rule but there are only four of these exceptions in which the doctor will ordinarily be interested.

One of these is this same rule as to pedigree. If a patient who later died told the doctor that X is her illegitimate son the statement will be admitted.

Another exception is the fact that on scientific matters on which the witness is called to testify he need not have acquired his knowledge through experience. He may have acquired it from books.[15]

The third exception is that where the doctor has obtained from others the information on which he based, in part at least, the treatment administered, he is generally permitted to testify as to such statements made to him. These statements may consist of three classes:

(1) Statements made by a patient to a doctor as to his present symptoms or exclamations of the patient showing present pain. There is no question about the admissibility of these statements.

(2) Statements by a patient to a doctor as to past symptoms. On this the courts are not in agreement, although the modern tendency is to favor admissibility.

(3) Statements made to a doctor by third persons. Here, again, the courts are not in agreement, but where the information comes from an attending nurse or another physician or from the wife or some other member of the family having personal observation and an interest in learning and describing accurately, there seems every reason for admitting testimony based in part on these statements.[16]

Dying Declarations

The fourth exception to the hearsay rule that the doctor will encounter is the dying declaration. Early in the history of the law of evidence it was decided by the courts that a statement made by a man about to face his Maker had such a ring of truthfulness about it as to warrant the court's receiving it even though a second hand report of the statement by one who heard it made is hearsay in nature.[17] The courts have seen fit to limit the application of the rule to but one class of cases, i.e., criminal prosecutions for homicide where the death of the declarant is the subject of the charge, but in those cases the rule is generally applied. Before the dying declaration can be admitted, it must be proved that the declarant, at the time that he made the statement, believed that he was going to die and the statement made must be one of fact and not of opinion. Therefore a doctor who is to listen to a dying declaration by a person suspected to have been injured by violent means so that he is in danger of death should take pains, by questions addressed to the patient, to ascertain, before the dying declaration is made, that the patient really believes that he is going to die. And, when the dying declaration is made in the form of an opinion or conclusion, questions should be asked by the doctor to bring out the facts that are the basis for the conclusion.

For example, as a dying declaration may be used to absolve a defendant from the charge of crime as well as to convict him, assume the following conversation between doctor and patient:

Patient, "I want to tell what happened."

Doctor, "Why, do you believe that you are going to die?"

Patient, "Yes, Bill shot me but it wasn't his fault." ("Not his fault" is a conclusion.)

Doctor, "Why wasn't it his fault?"

Patient, "He was shooting at a rabbit and didn't see me so near."

Going back to the description of the trial, when the party calling the witness concludes his examination he turns the witness over to the opponent for cross-examination. When the cross-examination is concluded the party who called the witness may again interrogate him to clear up matters brought out in the cross-examination. This is called "redirect" examination. When the redirect examination is concluded, the witness is through and may leave the stand. Some attorneys, however, are very cautious and selfish in insisting that the witness stay around as they may wish later on to ask him some more questions. If such a request is made and the doctor is a busy man who wishes to get back to his patients, he should not hesitate to tell the judge how busy he is and ascertain if the judge will not see to it that the witness is reëxamined right then and there or else released from attendance.

Qualification of Doctor as Expert Witness

As pointed out in the opening of this paper, there are three capacities in which the doctor may be called to testify: (1) as an ordinary witness who saw something happen, (2) as the doctor who treated the patient, (3) as an outsider called in to give his opinion on a problem of science. Capacity No. 1 presents no problem other than those that confront any witness. In Capacity No. 2 there is every expectation that the doctor will also be called upon to testify in Capacity No. 3. Therefore the attorney who calls the doctor will, right at the beginning, plan to qualify him as an expert. This will be by a series of questions planned to draw out the intellectual and scientific training of the doctor and his experience, if any, in the particular field under inquiry. These questions will be somewhat as follows: Please state your name? Where do you reside? What is your profession? What has been your general education? What medical school or schools have you attended? In what hospital did you interne? How long have you practiced medicine? Where? What has been the nature of your practice? Are you now connected with any hospital? Have you ever had any experience in treating fractured bones? What has that experience been? Do you belong to any medical or scientific societies? What are they?

Sometimes the attorney will simply ask the doctor a few preliminary questions and then ask him to state his qualifications as a medical man. The writer considers this practice unwise. A glib answer, reciting a long list of schools attended, membership in learned societies, breadth of practice, etc., will practically always place the witness on the defensive with the jury, as

being patently a man who makes his living by giving expert testimony, "an old hand at the game," to be carefully watched. It is much better for the attorney's case and for the appearance of the witness in the eyes of the jury to have these facts drawn out of the witness by a series of questions.

In this examination (which is called an examination on *voir dire,* being an examination to determine the qualifications of the witness as an expert) the witness should take pains not unduly to stretch his qualifications as an expert, for he may be cross-examined on that point. If he is a young practitioner who never set a leg in his life until he was called to this particular patient, let him not claim to be an experienced orthopedist. If asked as to whether he has treated similar cases, let him not answer, "Yes, dozens of them" for 24 is quite a large number and when the cross-examiner begins to ask him to state just what particular cases of this kind he did treat, when he gets through he may be able to recall only 5 such cases and that is embarrassingly less than 24.

On the other hand, of course, the doctor must not be too timid and modest about his qualifications. He should give the impression of a man who knows his profession but has no desire to brag about his qualifications or unduly exaggerate them.

Method by Which Expert Witnesses Are Examined

Let us first discuss the testimony of the doctor in Capacity No. 2, the man who treated the patient. After describing his diagnosis and the treatment which he administered, he may be asked the question, "From your knowledge of medicine and your experience in this case, what is your opinion as to the present condition of the plaintiff being permanent?" Doctor: "I believe that the condition will be permanent." Question: "What reasons do you have for such opinion?" The witness will then state the reasons which led him to the conclusion reached.

When, however, the witness testifies in Capacity No. 3 as an outsider who never treated the patient but who has been called as an aid to the court in determining scientific principles, or scientific conclusions from facts, he must be examined in a rather unusual manner, by being asked to answer one or more hypothetical questions based on some evidence already offered in the case. The following is an example of such a question:

"Let use assume, Doctor, a man of thirty years of age, who had prior to June 8, 1932, enjoyed good health and was physically strong, and that on the morning of June 8, 1932, he was seated in the back seat of an automobile traveling along a public highway at about twenty to twenty-five miles an hour, and that an automobile turns into the road upon which this car is traveling, and collides with the rear of the car in which the plaintiff is riding, and that he is thrown from his seat in the rear of the car up against and through the glass windshield, and receives a V-shaped cut on the left side of the face running in a position from the nose downward and then upward toward the ear, and that a splinter of steel pierces the spine at or near the base; that severe cuts and wounds are received on the chest and legs; that the plaintiff, bleeding

profusely from the head and legs, is taken to a hospital; that he was unconscious for six hours, that upon examination the next morning by a doctor he is discovered paralyzed in both legs, that there is no sensation whatever in his left leg; that there was a total lack of lines of expression upon the left side of the face and very slight mobility upon the right side of the face; and that he had exaggerated reflexes.

"Now assuming those facts, can you state with reasonable certainty what in your opinion was the cause of the plaintiff's paralyzed condition?" [18]

The reason for the use of the hypothetical question is apparent. The witness, knowing nothing about the facts of the case except by hearsay, can testify only as to the scientific conclusion which he is able to reach from the facts submitted to him. In giving his answer the witness must confine himself to the facts stated in the question. He cannot base his conclusion on those facts plus something else which he heard some witness say. Very often the facts on which the hypothetical question is based will be in dispute. In that case the opposing attorney, on his cross examination of the witness, may state the facts as narrated by his own witnesses and ask what, assuming such facts to be true, would be the opinion of the witness. To that question the witness may give as an answer an opinion exactly the opposite of his opinion given on the direct examination.

There is only one short cut to avoid the method of examination of an expert by hypothetical question. That is to have the witness on hand when another witness or other witnesses are telling their story and then ask the expert:

Question: "Did you hear the testimony given by Doctor X (or by Doctors X, Y and Z)?"

Witness: "Yes."

Question: "Assuming everything to which he (or they) testified to be true, what would be your opinion, etc.?"

Occasionally an inexperienced attorney will try this kind of a short cut:

Question: "Have you listened to all the testimony in this case?"

Answer: "Yes."

Question: "Based on such testimony, what is your opinion, etc.?"

Such a question is clearly objectionable as the testimony is probably in conflict and an answer to the question would compel the witness to decide which story to believe, which is not the function of the witness. Nor when his answer comes in will the jury know on just which of the disputed facts the opinion expressed by him was based.

Use of Memoranda While Testifying

The law expects the witness to tell the court what he now remembers, not what he has written down in advance as his testimony and which he may undertake to read to the court. The law realizes, however, that there may be lapses in memory and concedes that a witness may be allowed to have his memory refreshed. This can be done in two ways. One is called "present recollection," the other "past recollection recorded."

In what is called present recollection the witness cannot remember something that has occurred. He knows it but it is dormant in his memory. Something which he sees, usually something handed to him for the purpose, it may be a hospital record, it may be an office diary, it may be a prescription written at the time, it may be a report written to an insurance company, something pulls a switch in his brain and the memory of the whole thing comes back to him so that he can lay down the paper and tell the whole story out of his own memory. This "pulling the switch" is what is called "refreshing the memory" or "present recollection."

On the other hand, at the time of the occurrence the witness may have made a written memorandum or he may have at the time read a memorandum made by someone else. He does not remember at all what the contents of the memorandum were. An examination of it fails to pull any memory switch. He does know, however, and is willing to so swear that he remembers making the memorandum or reading the same and knows that the facts stated therein were at the time true. In such cases the law permits the memorandum to be introduced into evidence based on the above evidence of the witness. This is what is called "past recollection recorded."

There are some situations, however, where the witness may wish to refer to a document and it does not refresh his recollection nor can it be established as past recollection recorded. Can he use the document for any purpose? Yes, if it has already been introduced as evidence in the case.

For example, take a hospital record. There has been a great deal of discussion by judges and law writers as to whether these records are admissible in evidence. The courts are now pretty generally agreed that they are admissible when properly authenticated as business records. (That the hospital record shall be properly authenticated is a problem for the lawyer, not the doctor. The doctor, if he wishes to use the record in giving his own testimony, need only notify the attorney of his desire to have the hospital record for use in connection with his testimony to be given. It will be up to the attorney to obtain the record and to have it properly authenticated and admitted in evidence.) Once the hospital record is in evidence the doctor witness may refer to it in his testimony with perfect freedom, for he is only discussing a document that is already before the court as evidence in the case. *But the hospital record must have been admitted in evidence before the doctor undertakes to use it in his testimony.* If the attorney has not yet introduced it in evidence he should do so before the witness undertakes to testify about it. The same rule will apply as to the use of books of account and other records, including public records, death certificates, birth certificates, etc.

Use of Graphs, Charts, Diagrams and Photographs

To illustrate or confirm his testimony the doctor witness may use graphs, charts, diagrams and photographs. But these documents derive their competence as evidence altogether from the fact that they are given in connection

with the testimony of a witness. Such a document, offered without an identifying witness, would not be admissible.

The following are examples of the methods by which such documents are introduced in evidence.

Question: You have described to us the relative positions in which the bones appear in a normal body and the position in which you found them to appear on your first examination of the plaintiff. To illustrate your statements in those respects have you prepared any diagrams?

Answer: I have.

Question: Will you produce them? (The diagrams are produced and marked by number as exhibits for identification, viz., so that the record will show by exhibit number what is being discussed on the examination of the witness.)

Question: I hand you plaintiff's Exhibit No. 7 for identification and ask you what it is.

Answer: It is a diagram prepared by me showing the position in which the ———— and ———— bones appear in a normal body.

Question: I hand you plaintiff's Exhibit No. 8 for identification, and ask you what it is.

Answer: It is a diagram prepared by me showing the position in which I found the ———— and ———— bones of the plaintiff to be when I examined him on July 1, 1943.

Question: Are these diagrams correctly drawn?

Answer: They are.

Question: Do they properly show what they purport to show?

Answer: They do.

Attorney: I offer in evidence plaintiff's Exhibits 7 and 8 for identification.

For the introduction of a photograph the procedure might be as follows:

Question: You have testified as to the location of the railroad crossing and the location of the building which you have stated would obstruct the view to the north of one approaching the crossing from the west. I hand you plaintiff's Exhibit No. 11 for identification and ask you what it is.

Answer: It is a photograph of the railroad crossing about which I have testified taken from a point directly west of the crossing and showing the road, the railroad track, and the building on the north side of the road about which I have testified.

Question: Is this photograph a correct representation of the scene which it undertakes to describe as you remember that scene?

Answer: Yes.

It is apparent, from the theory that the chart, diagram or photograph is based on the testimony of the witness, rather than the contrary, that it is altogether immaterial by whom the chart or diagram was made or the photograph taken. The chart or diagram may be taken from a medical book, or it may be one prepared for instructing in a physiology or anatomy lecture.

The photograph may have been taken by any professional or amateur photographer. They all depend upon the credibility of the witness whose testimony they illustrate.

Although it is not necessary that the person who took a photograph be produced if the witness can sufficiently establish its correctness, it is, of course, well to have the photographer present to testify himself as to the manner in which the photograph was taken. That is particularly true where the photograph is of any part of the human body for the angle at which the photograph is taken may make a considerable difference in the effect to be given to the photographic representation.

Roentgen-Ray Photographs

Here are encountered problems much different from those that exist in the case of an ordinary photograph. For the proper reading of the roentgen-ray photograph depends so much upon angle and focus that the doctor witness cannot reasonably testify thereto until he has learned the conditions under which the roentgen-ray photograph was taken. Therefore the courts have had to lay down the following rules with regard to such photographs:

1. The person who took the roentgen-ray photograph must testify as a witness, (a) as to his qualifications, by training and experience, to take such photographs; (b) as to the trustworthiness and dependability of the instrument that was used; (c) as to the manner in which the photograph was taken, the position of the patient, etc.; (d) as to the identity of the photograph with the person to illustrate whose physical condition the photograph is offered.

When the doctor witness has taken the roentgen-ray photograph himself, he is the only witness needed. Where, however, the photograph is taken by another physician or by a dentist or by a technician, the person taking the photograph must appear and testify as to the above matters.

2. After the taking of the photograph has been thus established and the identity of the patient determined, it becomes the subject of the testimony of the doctor witness as to the proper interpretation to be placed upon it. For the law is well established that a roentgen-ray photograph cannot speak for itself. It must be interpreted by some person qualified by training and experience to make such an interpretation. Therefore the doctor who is to testify as to the findings established by the roentgen-ray photograph must not only qualify himself as a physician but as an interpreter of roentgen-ray photographs. He can have acquired such expert knowledge either by education or experience. The ordinary course in roentgenology given in medical schools will be sufficient to qualify the doctor although the more experience he has had in reading roentgen-ray photographs the more effective will his testimony be.

Suppose that the roentgen-ray photograph, after having been originally studied by the doctor, becomes lost? Can the doctor witness testify as to what the photograph showed? Although the courts are not altogether

agreed on that point, in most jurisdictions the doctor will be permitted to testify on such subjects after it has been proved (1) that the absent photograph is or was accurate, and (2) that it cannot be produced in court either because it is lost or is otherwise unobtainable.[19]

Motion Pictures

Motion pictures may be offered in evidence under either of two sets of circumstances, namely: (1) To show something that actually occurred. For example, when a motion picture is taken, without the principal actor's knowledge, to expose an alleged malingerer by showing him walking in an ordinary manner. (2) To show an attempt to reproduce a past scene by having the original characters therein, or persons representing such characters, go through the motions claimed to have taken place at the time of the original scene.

It may be pointed out in the first place that judges are generally opposed to the use of motion pictures in a court trial, for several reasons: (1) They take up considerable time in the making of arrangements for the showing. (2) They divert the attention of the jury from matters which are really of more importance in the case. (3) They tend to exaggerate in the minds of the jury the facts which they are offered to prove. Also, under the circumstances involved in No. 2, where persons act out a scene that has occurred in the past, the uncertainty of the accuracy of such acting, particularly where there is a sharp conflict in the testimony as to just what did occur at that time, makes the reception of such evidence very dangerous.

It may therefore be said that rarely, if ever, will the courts permit the use of such evidence for the purpose described in use No. 2.

In the first set of circumstances, however, where the camera records what actually did happen, the judge is likely to admit the evidence, particularly if he is convinced that otherwise a fraud might be perpetrated on the court by a malingerer. Often, before the judge will make his ruling on admissibility, he will have a private showing of the film in his chambers, by which view the judge can see the importance of the evidence offered, its application to the issues in the case on trial and its apparent reliability.

Going back to the original proposition that a photograph is admissible only in connection with the testimony of a witness, either to aid in understanding the testimony of such witness or as confirmation of the testimony given, it is apparent that a motion picture is subject to the same rule. A witness testifies as to what he saw. He then testifies that the motion picture is a reproduction of what he saw.

Also, before the motion picture can be introduced in evidence it must be authenticated as a true reproduction of the scene that it depicts. This authentication need not be by the photographer himself, anymore than in the case of a still photograph. But it is the sounder practice in the case of a motion picture to have it authenticated by the photographer who took it,

who should testify as to the manner in which it was taken, the speed in frames per second, etc., as a motion picture will not reflect accurately the movements of the subject unless the projection machine in the courtroom is operated at the same rate of speed as was the camera,[20] and that the picture is a correct reproduction of what the witness actually saw.

Preparation, by the Doctor, for the Trial

When the doctor is informed that he is to be called as a witness, it will be incumbent upon him to prepare himself for the examination.

If he is to be called as having treated the patient he should consult his notebooks and diary to see what he has written down regarding the case and should search his memory as to his first diagnosis and any later diagnoses, the treatment he administered, the progress of the patient under such treatment, etc. If the patient was taken to a hospital the doctor should ask to see and should study the hospital record of the case.

Inasmuch as he will also doubtless be asked to testify as an expert to give his prognosis as to the ailment treated, he should also make a careful and thorough study in his library of the medical literature on that particular subject, not only refreshing his memory on what he at one time learned but bringing himself up to date on all the modern developments in that field of medical knowledge. If his testimony is to be based at all on his own experience in treating such ailments, the doctor should look up his old notebooks and diaries so as to prepare himself for either direct or cross-examination on the extent of his experience in that field and the lessons he learned therefrom.

Study to Simplify the Use of Medical Terms

Theoretically, and practically, the expert witness is there as an aid to the judge and jury in advising them of the scientific problems involved in the particular case and in helping them to apply the rules of science to the facts in that case. He can be of real value only if he can talk to the judge and jury in terms that they as laymen can understand. Unfortunately, in the average case, the medical testimony comes out in the form of a technical jargon that is almost completely unintelligible to the jury and can only be translated to them by the judge in his charge or by counsel in their arguments if they themselves understand the language used.

Sometimes this manner of testifying comes from the vanity of the witness in wishing to display his erudition. Generally, however, it is because the expert uses the language of the books and that used by him in discussing medical cases with his colleagues. It simply does not occur to him that these words are not perfectly intelligible to everyone.

The writer urges, therefore, that every doctor who is to be a witness go over his testimony in advance with the thought in mind as to each medical term used, "Is this term intelligible to the ordinary layman? If not, can I

express the same thought in language that the layman will understand even though I may not be able to convey the exact shade of meaning that I might try to obtain by the use of the more technical term?"

For example, the doctor will ordinarily be inclined to use the word "trauma." Cannot he convey substantially the same meaning by saying "injury?" Instead of "ecchymosis" cannot he say, "He had a black eye." Instead of using the technical terms to describe the operation cannot he say, "I turned back the scalp and exposed the skull."

So also, cannot the doctor express, in words more easily understood, such medical terms as ankylosis, axilla, ligation, osteitis, palpation, posterior, reduction, scapula, sternum, thorax, hypertension, etc.

If the doctor cannot think of the layman's term for a certain bone, organ or bodily condition, he can often obtain valuable help by looking up in his medical dictionary the term he plans to use and seeing whether, in the definition given, there is not some short term known to the layman.

If the doctor witness feels that no term intelligible to laymen will give the exact meaning of the technical term that the doctor would like to use, he can use his technical term and then attempt to translate it for the jury, "It is hard to define that condition by any less technical word than the one that I have used. It means ————————. Very roughly you might use the term ————————."

If the attorney has properly prepared his case he will have gone over the doctor's testimony with him before putting him on the stand. In that interview the doctor can call the attorney's attention to certain medical phrases he expects to use and see whether or not they will be intelligible to the most important laymen in the case, the judge and the jury.

The Direct Examination of the Doctor Witness

If the attorney has properly gone over with the witness the testimony he is to give, the latter will know very well what questions will be asked him on direct examination and will have thought out the replies that he will make to such questions. The principal advice to be given him is to listen carefully to each question asked and not to undertake to give his answer until the attorney asking the question has finished and the witness is sure that he understands the question. If there is anything about the question that he does not understand he should not hesitate to have the question repeated. If he still does not understand it he should frankly say so and afford the attorney an opportunity to so reframe the question that it will be intelligible to the witness. If in a hypothetical question there is any fact missing that would be of value in formulating the opinion requested, the witness should say, "I could answer that question better if I knew so and so." If the missing fact can be supplied by the interrogator from the testimony in the case he will supply it. If not, and it is impossible to give a trustworthy opinion without the missing fact, the witness should not hesitate to so say.

Cross-Examination

This, unfortunately, is something that is looked forward to by every inexperienced witness as an ordeal. He has heard or read so many stories of witnesses being broken down on cross-examination that he fears the same thing will happen to him.

To reassure the doctor witness that most of his fears are unfounded, the writer will quote from an attorney whose function it has been to try to break such witnesses down. In writing a treatise for attorneys on the subject of "Medical Trial Technique," the author, an attorney of great experience in the trial of cases, says, in respect to cross examination of the medical witness:

"The more experience one has in the cross-examination of the medical witness and particularly the medical *expert* witness the more one must come to the conclusion that the cross-examination of a truthful, honest, efficient, and capable medical expert witness who is not given to exaggeration is not only dangerous but usually harmful to the trial lawyer. When this type of a medical witness is encountered, it is no wonder that the most experienced and most successful trial lawyers in personal injury cases frequently make the statement that the best cross-examination of such a witness is no cross-examination. It is only when the witness is neither truthful, honest, efficient, nor capable—and is given to exaggeration that one can expect to successfully destroy a witness' story by cross-examination." [21]

If the opposing counsel does decide to cross-examine it may be for any one of numerous reasons.

He may decide only to weaken the testimony of the witness in the eyes of the jury by showing that he has an interest in the case, e.g., that he has a large bill for attendance on an indigent patient which bill will never be paid unless the patient recovers damages in this suit; or that he is being paid a large fee for testifying; or that he is a "professional" expert spending a large amount of his time in court as an expert witness in actions for personal injuries or in will cases if the doctor is a psychiatrist or neurologist.

The attorney may attempt to make the witness admit one or more facts or one or more matters of opinion which will tend to corroborate the examiner's theory of the case.

He may attempt to show that there were certain omissions in the testimony of the witness as to the physical condition in issue, the examination by the doctor, the diagnosis, the treatment, or the prognosis.

If the doctor witness is honest, able, and truthful, he has nothing to fear from any such lines of inquiry. The things for which he should be on the watch are as follows:

1. Confusing questions, i.e., questions that are indefinite and uncertain, double questions, questions that assume the truth of a fact not yet proved.

For example, a question, "If the car were traveling partly in the right lane and partly in the middle lane of the three car highway, how much space would that leave for the cars to pass?" would be indefinite and uncertain. It could not be answered without knowing (a) on which side the other car was to pass, (b) how far into the middle lane the first car was traveling.

A question, "Was the wind from the northeast and of gale strength?" would be a double question, two questions in one. "Was the wind from the northeast?" "Was it of gale strength?" The questions should be separated before the witness should be called upon to answer.

The question, "What part of the plaintiff's body were you examining when you found indications of the former injury?" may be objectionable as assuming a fact not yet proved, viz., that you, the witness, had found in the patient indications of a former injury.

The attorney who called the witness to the stand should be on the lookout to see whether a cross-examiner's question is confusing and to object thereto. If he is neglectful of his duty in this respect the witness should say, "I cannot answer that question."

Question: "Why not?"

Answer: "It is indefinite and uncertain," or "It is two questions in one," or "It assumes that I did find indications of a former injury and that is not the case." The judge will then compel the cross-examiner to reframe the question.

2. The next thing for which the cross-examined witness should be on the watch is some former opinion on a medical question given by the witness which is not in accordance with the theory on which he now testifies. The opinion may be in a book written by the witness or in an article by him in a medical journal. It may be some testimony that he gave years ago as a medical expert in another law suit. If the opinion that was given at the former time was then believed to be the truth but the witness has since changed his mind, he should not hesitate to so state, "Yes, I wrote the article referred to. It was my opinion at the time. I have since changed my opinion on that point and if I were to rewrite that article (if the witness did rewrite the article and can cite the new article to the court he will have scored a triumph) I would report that change in my views." The cross-examiner will doubtless then ask, "What caused you to change your views?" (If the cross-examiner does not ask that question the attorney who called the witness should take pains to ask it on his redirect examination.) Answer: "Further study and experience by me in that class of cases and the writings and researches by others along the same line."

3. A third line of attack for which the witness should be prepared is an attempted trick of a cross-examiner to persuade the witness to cite, as the basis for his opinion, an authority who never existed or an alleged statement by an authority who did exist but who never made the statement claimed. For example, after the cross-examiner has had the witness give the names of certain authorities supporting his theory, he may ask (often with a pile of what look like medical books on the table in front of him):

Question: "Are you acquainted with the writings of Dr. ————————?"

Answer: "Yes."

Question: "Of Dr. ————————."

Answer: "Yes."

Question: "Of Dr. ————————."
Answer: "Yes."

naming in his list of writers at least one man who never existed. Or the questioner may refer to a book by an authority who did exist but who never wrote any such book. Or in an extreme case (extreme for it would be perpetrating a fraud on the court), he may attempt to read from a book something which is not there and have the witness agree or disagree with such statement.

Of course there are only two classes of witnesses against whom the above technics might ever prove effective. One would be the vainglorious charlatan who affects a learning that he does not possess. The other would be the inexperienced young doctor with an inferiority complex who is afraid to admit that there is anything that he does not know. The way to meet such a cross-examination is for the witness, when an author's name is stated or a work is stated of which the witness has never heard, frankly to say that he knows of no such author or no such work. If the questioner undertakes to read from a book let the witness ask to see the book.

Manner of Testifying

What makes one doctor a good witness and another a poor one? The difference is more than in the amount of learning possessed by one or the other or the amount of experience that either has had. It is in the impression he makes on the jury as being earnest, as being an able scientist, and as being able to explain difficult matters to a jury in a manner and in language that they will understand. The following advice can be given to a doctor who desires to be an effective witness.

1. The doctor should be honest with his client and with the court. If the client's injuries are trifling and there is very good certainty of recovery he should be so told. If the sound consensus of medical opinion is against the contentions of the party by whom he is called, he should so tell the attorney calling him and if the attorney persists in placing him on the stand he should so tell the court.

2. The doctor should not undertake to testify as an expert witness unless he feels that he is qualified on that particular subject. If he has spent his whole life in general surgery he should not testify in a will case on problems involving mental diseases, unless he has as a side line interested himself in such problems, anymore than the ordinary psychiatrist should testify as to the best method of reducing a Potts fracture.

3. If asked a question that stumps him the doctor should not hesitate to say, "I do not know the answer to that question."

4. The doctor should not be too "cagey" in his replies to questions, for he may give the impression of either not knowing the subject on which he is examined or of not being willing to give his opinion on anything.

5. He should attempt to make a direct answer to every question asked

him. If he thinks a direct answer "Yes" or "No" might be misleading, he should so state to the judge.

6. He should know and remember that he is there to help the court to a better understanding of the problems under consideration and that no matter how hard a cross-examiner presses him he will always have the opportunity to explain his answers to questions. He may say at the time, "I think that I should explain that answer." Generally the judge will let him do so. If not, he can explain it when his redirect examination is conducted by the attorney who first called him to the stand. If this attorney overlooks calling for that explanation the doctor should remind him of his desire to so explain.

7. He should depend on his own knowledge of the questions of science involved. In other words, he should not ordinarily quote from medical books unless the cross-examiner insists on his so doing. The cross-examiner may confront the witness with a quotation from a medical book but if the doctor witness considers that the book is wrong and he is right he should not hesitate to say so.

8. He should take care not to use expressions that will make his testimony seem indefinite, "I think," "My impression is," etc. If he really is in doubt he should say so, otherwise his statements should be firm and direct.

9. If his testimony is to be given in narrative form, following such a question as, "when you first examined the patient what did you find his condition to be?" the story that the witness is to give should be arranged in a logical order that can easily be followed by the jury and should be given without digressions or retracings.

10. He should speak slowly (though not too slowly, for that would bore the jury), audibly and distinctly, turning his face to the jury rather than to the examining counsel and address his testimony to the jury. His words and manner of speaking should be just as if he were explaining in his office the same matter to some layman who was interested in learning just where the truth lay.

11. It is needless to say that the doctor should never argue with counsel, also that he should never lose his temper.

Testimony in Malpractice Cases

There is a rule of evidence (which is really a part of the substantive law of malpractice) that, ordinarily the want of skill or care on the part of the doctor can be proved only by expert witnesses. The reason for the rule is obvious. As the plaintiff's case is based upon the contention that the doctor failed to use proper skill or care, the question of whether proper skill or care was used must be determined by those who know what proper skill or care should be. No doctor should be mulcted in damages just because the patient does not now feel well. Although there may occasionally be a case where the negligence of the doctor was so gross that the court will dispense with this rule, in most cases it will be rigorously applied. The result is

that in a suit for damages for malpractice, no matter how meritorious the cause of action may be, if the plaintiff cannot find a doctor who is willing to testify in his behalf it will be impossible for him to prove his case. Often this will be so. All the doctors in the community, with a feeling of mistaken loyalty, may take the position that they must line up for the defendant whatever the merits of the case may be.

The doctor owes a duty to his fellow practitioners to see that the latter are protected against fake causes of action. On the other hand, where there has been actual and actionable negligence on the part of the doctor, the other members of the profession owe a duty to the cause of justice to see that the victim of the malpractice is properly compensated. Let it be hoped that a sense of justice rather than that of mistaken loyalty will guide the decision of the doctor who is asked to testify in such a case.

Extra Compensation for Expert Witnesses

As was stated earlier in this paper, it is the duty of any person who knows anything about the case, including a doctor who treated a party to the cause, to attend the trial when summoned by a writ of subpoena and for such attendance he is entitled only to the ordinary fees prescribed by statute. When, however, he is called as an expert, not because he knows anything about the facts in this particular case, but because he is needed to advise the court upon a principle of science involved in the case, is he entitled to be paid on a professional rather than on an ordinary witness basis?

This problem has occasioned much discussion in the courts, resulting in a considerable division of authority. The argument is made, on the one hand, that the exaction of the valuable special services of an expert, without other than the ordinary witness' pittance is a hardship that ought not to be imposed. On the other hand, it is argued that the hardship upon the professional man who loses his day's fees of fifty or a hundred or more dollars is no greater relatively than upon the clerk or mechanic who loses his day's earnings of five or ten dollars;—each loses his all for the day. Each owes his duty as a citizen to attend and give his evidence when summoned by the court. The same loss of income would occur to the doctor if he were summoned to be a juror and yet he would never think of demanding extra compensation for his services in that public capacity.

Decisions of the courts of different States will be found holding either way on this question and the legislatures also have stepped into the discussion by enacting statutes on the subject. Some such statutes provide for extra compensation for expert witnesses. Other statutes expressly forbid such payments. If the doctor wishes to stand on his rights in that regard, he must investigate to see what the statutes and decisions are in his particular jurisdiction.[22]

Fortunately, however, there is a practical side to this problem which generally renders a study of statutes and decisions unnecessary. The party

calling an expert witness will generally expect to pay him extra for his services, just as he would expect to pay a workman whom he has caused to be summoned an extra amount equivalent to his "lost time." Why should he make such payments when he can obtain the services of the expert for only the normal witness fee? Is the testimony of the expert witness for sale to the highest bidder? Unfortunately history shows that in some cases the answer to the above question is "Yes." The testimony of some expert witnesses seems to vary in effectiveness in accordance with the size of the fee paid for their appearance. Theoretically, however, the compensation which is paid to an expert witness is not for his services as such, but to reimburse him for what he has lost by giving to a particular case time otherwise available for the remunerative practice of medicine. This, of course, is the same theory on which a doctor bases his charges for traveling to treat a patient in a distant city. Also, what the party desires when he summons an expert is not just the off-hand opinion of the witness given on the stand. He wishes an advance study by the prospective witness of the facts in the particular case and of the problems of science involved in the case. For the time spent by the expert in this advance study the party will expect to pay compensation. Therefore there is no reason ethically why a doctor sought as an expert witness should not inform the party calling him that he expects to be compensated for the time he devotes to performing the duties expected of him. Involved in this problem, however, is the question of the duty of the doctor to appear and testify without charge for an indigent litigant who is without the means to compensate his witness. That is a matter of individual conscience for the doctor himself to decide. If he were the one who treated the party as a patient there is little question that it would be his duty to see the case through by testifying on the patient's behalf. On the other hand, if he has had no professional relations with the party and feels that he is being called only because of his eminence in the profession, he may regard the call as an imposition, unless to decline it might, in his opinion, lead to a miscarriage of justice.

The amount of the compensation demanded should not be more than fair reimbursement for time lost based on the doctor's ordinary scale of fees. A greater charge would subject the doctor to the accusation that he is selling his testimony.[23]

Improvements in the Law of Evidence as to Expert Witnesses

It is one of the scandals of the law (as well as a scandal in medicine) that, in a case involving medical testimony a litigant can apparently always (except, as above stated, the plaintiff in a malpractice case) find some medical expert to support his theory of the case and that the testimony of medical experts varies with the necessities of the side by which they are called. That is why, for many years, there has been a strong movement, fostered by experienced judges and by the better class of lawyers, to have it that instead of

experts being called by each side to oppose one another in hostile array, each expert apparently owing a duty to the side by which he was chosen, the experts be selected by the judge, either by agreement of the parties or on his own motion—such experts to owe a duty only to the court, that duty being to determine just where the truth lies as to the rules of the science involved in that particular case and their application to the facts of that case. This movement has resulted in the drafting of the Uniform Expert Testimony Act, a statute prepared by the Commission on Uniform State Laws, which statute it is hoped will eventually be enacted in every jurisdiction.

This act provides that where, in a civil or criminal proceeding, issues arise upon which a court deems that expert evidence is desirable, the court on its own motion, or on request of either party, may appoint one or more experts, not exceeding three on each issue, to testify at the trial. These experts shall make such investigation of the subject matter as they deem necessary and shall make their finding in the form of a written report to the court. In addition to their report the experts may be called to testify by the court or by a party to the cause. The parties themselves may also call experts but must give reasonable notice of the name and address of the expert who is to be called and the jury shall be advised as to what experts were appointed by the parties and which ones were appointed by the court. An expert may be asked to state his inferences, whether they are based on his personal observation or on evidence introduced at the trial and seen or heard by the witness or on his technical knowledge of the subject, without first stating hypothetically in the question the data on which these inferences are based. The compensation of these experts appointed by the court shall be fixed by the court and shall be paid by the litigants in equal parts to the clerk of the court and thereafter assessed as costs of the suit depending upon which party wins.

The passage of such an act in every state will effect a greatly needed reform in the law of procedure in respect to those numerous cases where medical expert testimony is needed to guide the administration of justice.

NOTES

1. New York Civil Practice Act No. 352.

The reader's attention is drawn to the fact that in the Symposium on "Scientific Proof and Relations of Law and Medicine" (1st ser., 1943), Professor Zachariah Chafee, Jr. contributed a paper entitled: "Privileged Communications: Is Justice Served or Obstructed by Closing the Doctor's Mouth on the Witness Stand?" Yale Law Journal *52*: 607, June, 1943. The author pointed out that seventeen states . . . "seem to preserve the view of the English common law that there is no legal check upon the revelation of medical secrets," so that "On the witness stand, at all events, a doctor in these states must tell all he knows," these states being as follows: Alabama, Connecticut, Delaware, Florida, Georgia, Illinois, Maine, Maryland, Massachusetts, New Hampshire, New Jersey, Rhode Island, South Carolina, Tennessee, Texas, Vermont, and Virginia.

Professor Chafee also lists the states which have passed statutes of various sorts

similar to the New York law, and notes the exceptions to the general prohibition contained in the various enactments, saying: "The ensuing list mentions only the date of the original enactment without regard to subsequent amendments. The statutes vary in their terms, particularly as to waiver of the privilege. The ensuing list mentions only variations of especial medical interest, including the fact of adoption of the Uniform Narcotic Drug Act (U.N.D.A.): Alaska (1913) (except for insanity); Arizona (1913) (U.N.D.A.); Arkansas (1919); California (1872) (except for mental condition and venereal disease); Canal Zone (1934); Colorado (1921); District of Columbia (1919) (U.N.D.A.); Georgia (1935); Hawaii (1925) (U.N.D.A.); Idaho (1919); Indiana (1926); Iowa (1897) (U.N.D.A.); Kansas (1923); Kentucky 1915); Louisiana (1928); Maryland (1935) (U.N.D.A.); Michigan (1915) (except for illegal marriage of persons sexually diseased); Minnesota (1913) (except for bastardy); Mississippi (1906); Missouri (1919) (except for abortion); Montana (1935) (U.N.D.A.); Nebraska (1922) (U.N.D.A.); Nevada (1912) (U.N.D.A.); New Mexico (1929) (U.N.D.A.); New York (1828) (except for narcotic investigations); North Carolina (1919) (allows presiding judge of superior court to compel disclosure when necessary to administration of justice, U.N.D.A.); North Dakota (1913); Ohio (1921) (U.N.D.A.); Oklahoma (1931) (U.N.D.A.); Oregon (1920) (U.N.D.A.); Pennsylvania (1895); Philippine Islands (1901); Puerto Rico (1911) (except for malpractice, U.N.D.A.); South Carolina (1934) (U.N.D.A.); South Dakota (1919) (U.N.D.A.); Utah (1917) (U.N.D.A.); Virgin Islands (1920); Washington (1909); West Virginia (1897) (U.N.D.A.); Wisconsin (1919) (except for lunacy and malpractice, U.N.D.A.); Wyoming (1920) (U.N.D.A.)." Chafee, *id.*, pp. 607–8, f.n. 4.

He points out that . . . "several of the states recognizing the doctor-patient privilege in general have adopted the Uniform Narcotic Drug Act, which provides in sec. 17, par. 2, that "information communicated to a physician in an effort unlawfully to procure a narcotic drug, or unlawfully to procure the administration of any such drug, shall not be deemed a privileged communication." In a supporting footnote, he says: "This statute has been adopted in the following states and territories, of which those starred in the list do not recognize a general doctor-patient privilege: Arizona, District of Columbia, Hawaii, Iowa, Maryland,* Montana, Nebraska, Nevada, New Mexico, North Carolina, Ohio, Oklahoma, Oregon, Puerto Rico, South Carolina,* South Dakota, Tennessee,* Texas,* Vermont, West Virginia, Wisconsin, Wyoming." Chafee, *id.*, p. 608, f.n. 5.

2. In this respect the New York legislation is unique as rarely will such a statute be found to include nurses.
3. Wigmore, J. H.: Treatise on Evidence, ed. 3, Little Brown & Co., Boston, 1940, Vol. VIII, p. 817.
4. *Ibid.*
5. *Id.* at p. 818–9.
6. Battis v. Chicago, Rock Island & Pacific Ry. Co., 124 Iowa 623, 100 N.W. 543 (1904). A station agent called the company physician to examine a man who had been ejected from a train. Finding a small wound, the doctor dressed it. Held, he could not testify as to anything that the injured man said to him.
7. Wigmore, J. H.: *loc. cit. supra*, no. 3 at p. 823.
8. *Id.* at p. 824.
9. Green v. R. Co., 171 N. Y. 201, 63 N.E. 958 (1902). Here there was no claim that the information given was in any way necessary for the doctor to prescribe treatment. Situations can be imagined, however, where the story of how an injury occurred would be important in prescribing treatment, e.g., a wound apparently caused by a bite.
10. Perry v. Hannagan, 257 Mich. 120, 241 N.W. 233 (1932).
11. Wigmore, J. H.: *loc. cit. supra*, no. 3 at p. 831.

12. Wigmore, J. H.: *loc. cit. supra,* no. 3 at pp. 830–840.
13. *Id.* at p. 840.
14. *Id.* at pp. 808–810.
15. *Id.* Vol. III at pp. 2–4. The author quotes very appropriately from the great dramatist in Pericles, Act III, Sc. 2.

> "I ever
> Have studied physic, through which secret art
> By turning over authorities I have—
> Together with my practice—made familiar
> To me and to my aid the blest infusions."

16. *Id.* at p. 8.
17. See again Shakespeare, King John, Act V, Sc. 4.

> "Have I not hideous death within my view,
> Retaining but a quantity of life,
> Which bleeds away, even as a form of wax
> Resolveth from his figure 'gainst the fire?
> What in the world should make me now deceive,
> Since I must lose the use of all deceit?
> Why should I then be false, since it is true
> That I must die here, and live hence by truth?"

18. Schweitzer, S. C.: Trial Manual for Negligence Actions (2nd ed.), New York, Baker, Voorhis & Co., 1941, p. 829.
19. Scott, C. E.: Photographic Evidence, Kansas City, Vernon Law Book Co., 1942, p. 746.
20. *Id.* at pp. 506–514.
21. Goldstein, Irving, and Shabat, L. Willard: Medical trial technique, 1942, Chicago, Callaghan & Co., p. 19.
22. See, in this Symposium series, Goble, George and Smith, Hubert W.: Rights of Compensation for Medical Services.
23. Fee arrangements which are contingent on success of the litigant at whose instance the physician testifies, and agreements for a percentage of the damages recovered, are generally held by the courts to be illegal and so void. The reason is that any fee arrangement which gives the expert witness an interest in seeing one party prevail is inconsistent with his obligations to testify truthfully and impartially as an officer of the court. See Goble and Smith, *id.*

OBSERVATIONS ON MASS CHEMO-PROPHYLAXIS WITH SULFADIAZINE *

By Bennett W. Billow, M.D., *New York, N. Y.*, and M. S. Albin, Major, M.C., A.U.S.

It is the purpose of this communication to report an analysis of mass sulfadiazine prophylaxis. About 20,000 soldiers were given the prophylactic medication for a period of five weeks. Perusal of the literature reveals that the prophylactic use of sulfonamides has been investigated and studied by both military and civilian authorities. Thomas and France [1] administered prophylactic sulfanilamide for four years in doses of 1.0 to 1.2 grams a day to a group of rheumatic adolescents and young adults and compared the results with those observed in an untreated control group of similar subjects. Their results showed not a single major attack of rheumatic fever occurred in any patient, nor did any of the patients suffer from any acute beta hemolytic streptococcic infection. In contrast, 15 major rheumatic episodes were observed among the control patients during the same period. Coburn and Moore [2] gave prophylactic sulfanilamide to a group of rheumatic children and observed only one rheumatic recurrence among 184 subjects. Favorable results with prophylactic sulfonamides in preventing rheumatic recurrences have been published by Feldt,[3] Kutner, and Reyersbach,[4] etc. Thomas,[5] in summarizing the literature on rheumatic recurrences, states "that up to present time (October 1944) in civilian life prophylactic sulfanilamide has been administered to rheumatic subjects for a total of 815 patient persons over a period of seven years. Only eight have had recrudescenses, an incidence of less than 1 per cent, while the incidence among control groups ranged from 10 to 35 per cent." Investigations conducted during the past two years in the Army by the Army Epidemiological Board and others indicate that the incidence of meningococcal meningitis, of certain streptococcal diseases, and certain upper respiratory diseases of bacterial origin may be markedly reduced by the prophylactic administration of small doses of sulfadiazine.[6] The U. S. Navy's experiment last winter with mass sulfadiazine therapy resulted in an 80 to 90 per cent reduction in hospitalization for severe respiratory diseases, while streptococcic infections were reduced by 85 per cent and meningococcus meningitis practically disappeared.

Sick call records at our seven dispensaries during the first 15 days of January, 1945, revealed an unusually high sick call rate, the majority of patients exhibiting upper respiratory infections characterized by malaise, low grade fever, and red throats. Most of these patients improved with routine therapy and 24 hour rest in quarters. There was also a high percentage

* Received for publication September 14, 1945.

(23 per cent) of acutely ill patients with high septic temperatures, diffusely injected pharynges, and cervical and submental adenopathy, resembling clinically acute streptococcic sore throat. These were hospitalized. Further, during the four weeks preceding January 15, 1945 we had admitted eight cases of rheumatic fever.

During the eight day period ending January 7, 1945 the average number of daily hospital admissions for respiratory diseases was 9.7, about 7 per cent of which was attributed in origin to streptococci. In the following eight day period this daily average jumped to 37.5. Clinically, about one fourth of the 300 cases admitted during this latter period were of streptococcal origin. During comparable periods in 1944 we averaged 28.4 and 19.6 daily admissions, respectively. It should be noted that the trainee population at this post is almost completely renewed every four to five months. Each week approximately 1000 trainees leave camp and a like number arrive fresh from civilian life. The training cycle of 15 weeks thus represents practically the total military experience of our subjects in this study.

Army regulation [6] authorizes the use of sulfadiazine prophylaxis when the admission rate per 1000 per annum for common respiratory diseases including influenza exceeds 400 for a period of a week or more, provided that more than 20 per cent of the cases can be attributed to the hemolytic streptococcus bacteriologically or clinically. During the period of January 8 through 15, our hospital admission rate per 1000 per annum for common respiratory diseases jumped to 585; therefore, chemoprophylaxis was instituted. Reference to chart 3 reveals that this incidence of respiratory diseases was almost twice as great as during a like period in 1944. It will also be noted that we had had an even greater epidemic in December 1943 and January 1944 and we feared a repetition during the early weeks of 1945, inasmuch as the prevailing epidemiologic factors were essentially unaltered. These facts provided additional incentive for the institution of sulfadiazine prophylaxis.

In this chemoprophylaxis program one gram of sulfadiazine was administered daily to all infantry personnel for a period of five weeks. The soldiers were instructed to report to the dispensary immediately upon the discovery of a rash or any other untoward reactions to the drug, such as sore throat, nausea, vomiting, etc. The dispensary surgeons were likewise advised to be on the alert for toxic manifestations. In addition, the medical officers were directed to notify in writing each reactor's commanding officer, recommending immediate discontinuation of prophylaxis. A duplicate notification with added description of the reaction was to be forwarded to one of us (MSA). Thus, an opportunity was offered to the authors to investigate and study the results of mass sulfadiazine prophylaxis.

Effect of Prophylaxis on Hospital Admissions. Chart 1 shows the number of common respiratory diseases hospitalized daily from December 1, 1944 through March 31, 1945. From this chart it is evident that almost all lows occur on Sundays, and a few on Saturdays. It is, of course, a common

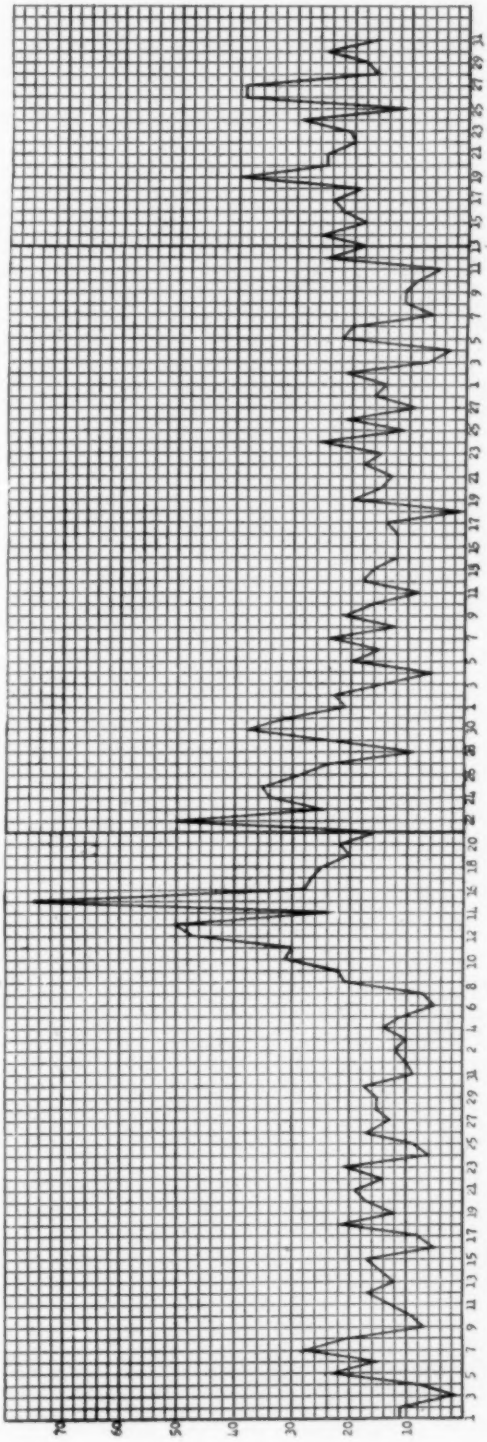

CHART 1. Daily hospital admissions for common respiratory diseases from December 1, 1944 through March 31, 1945.

observation that trainees are loath to report on sick call on Saturday evenings and Sunday mornings. It is, therefore, quite significant that on Sunday, January 14, 1945, twenty-two respiratory cases were hospitalized which, with but one exception, exceeded the admissions for any single day during

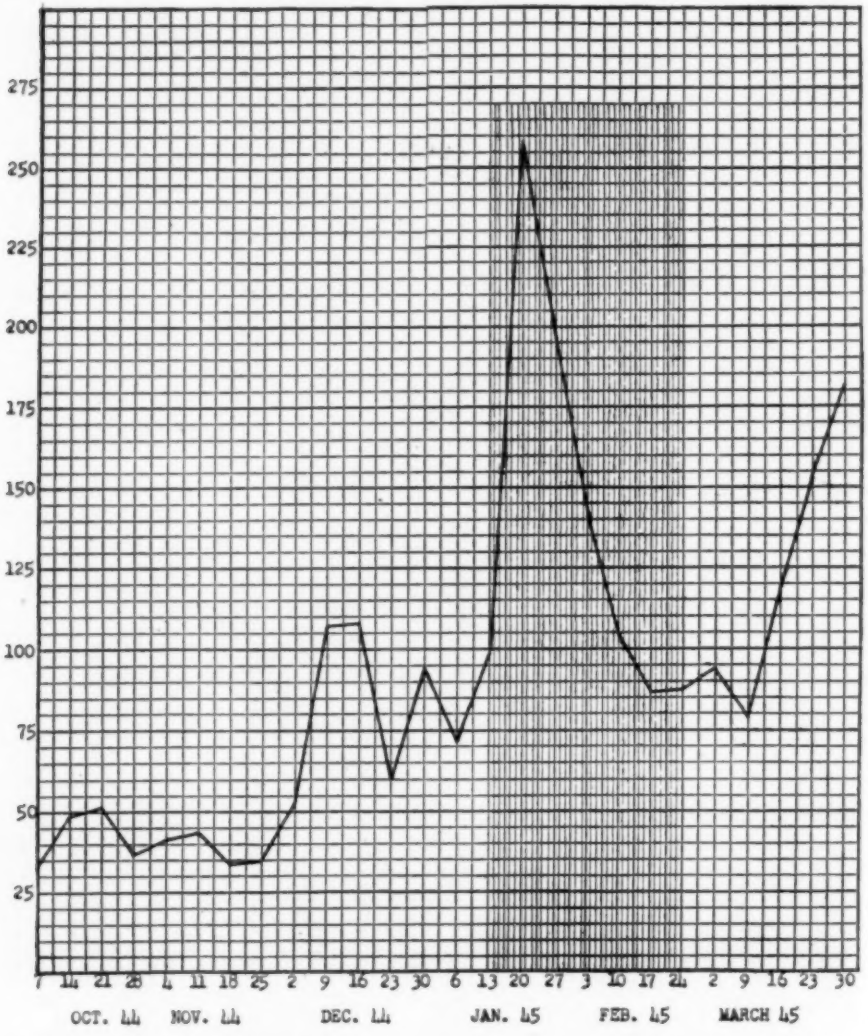

CHART 2. Weekly hospital admissions for common respiratory diseases from week ending October 7, 1944 through week ending March 30, 1945. Shaded area represents period of chemoprophylaxis (January 15, 1945 through February 24, 1945).

the preceding 15 weeks. It is evident, also, that there is a sharp progressive rise during the period January 7–13, then the Sunday recession, and then the peak of 75 cases on January 15. That afternoon mass chemoprophylaxis was begun and the incidence is seen to drop to 28, 27, 25, and 20 on

January 16, 17, 18 and 19, respectively. Sub-peaks are then reached on the following Monday and then on Tuesday of the succeeding week. Thereafter, admissions closely parallel those of the six week period prior to chemoprophylaxis. After discontinuation of prophylaxis on February 24,

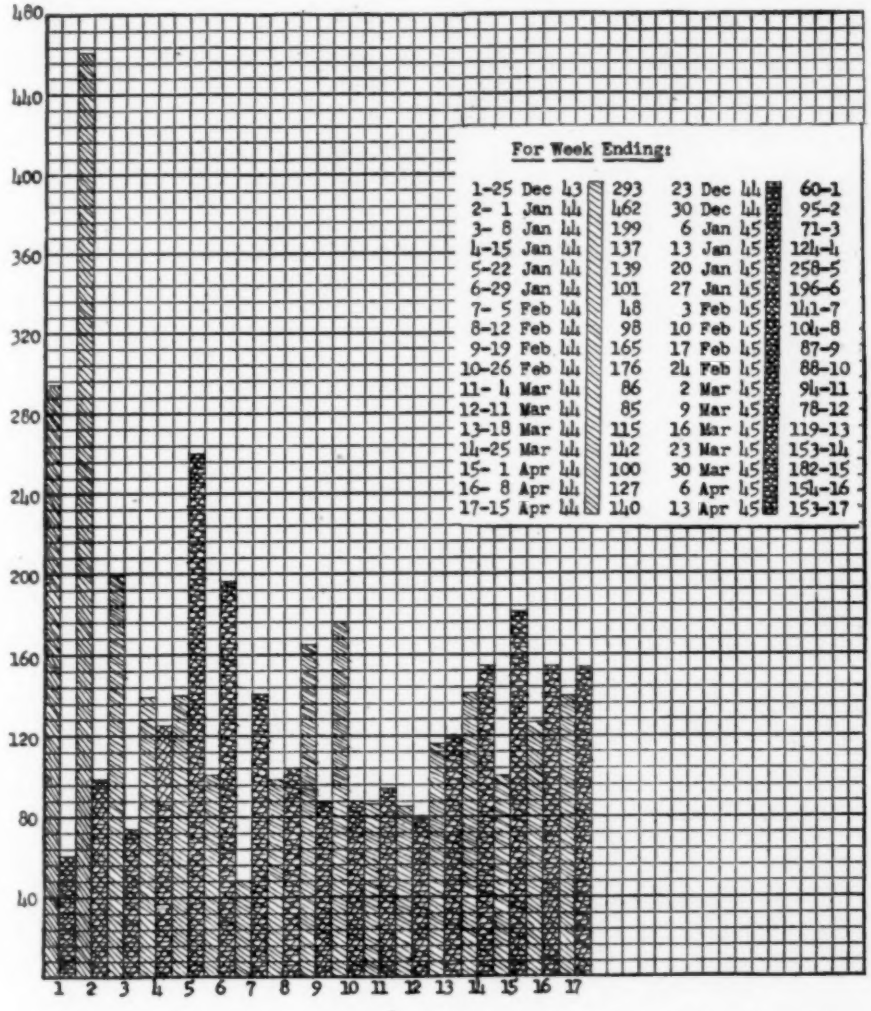

CHART 3. Common respiratory admissions to hospital by weeks in comparable periods of two successive years.

the incidence remains low until March 12, when the curve attains a higher level and continues to ascend.

Projected on a weekly basis, thereby contracting the daily figures of chart 1 sevenfold and absorbing the Saturday and Sunday lows, this respiratory curve is brought into bold relief by chart 2. Chart 2 illustrates the steady decline of respiratories during the period of prophylaxis, the apparent

after-effects of the program, and then the re-acceleration of the rate in March, 1945.

Table 1 contains an analysis of the total admissions to the hospital by weeks broken down into the following disease groups: common respiratory diseases, meningococcic meningitis, rheumatic fever, pneumonia, and scarlet fever. During the period of chemoprophylaxis from January 15 through February 24, no cases of meningitis (meningococcic) occurred. During the

TABLE I

Weekly Hospital Admissions for Common Respiratory Diseases, Meningitis, Rheumatic Fever, Pneumonia and Scarlet Fever
October 7, 1944 through April 13, 1945

For Week Ending	Common Respiratory Diseases	Meningitis (Meningococcic)	Rheumatic Fever	Pneumonia		Scarlet Fever
				Lobar	Atypical	
Oct. 7, '44	33	1	2	0	1	0
Oct. 44, '44	49	0	0	1	1	0
Oct. 21, '44	51	0	2	0	0	0
Oct. 28, '44	37	0	1	0	11	0
Nov. 4, '44	42	0	1	3	4	0
Nov. 11, '44	44	1	2	0	10	0
Nov. 18, '44	34	0	2	0	3	3
Nov. 25, '44	35	0	1	1	6	1
Dec. 2, '44	53	0	1	0	8	0
Dec. 9, '44	107	0	2	2	10	1
Dec. 16, '44	108	0	0	2	19	2
Dec. 23, '44	60	0	4	1	14	0
Dec. 30, '44	95	0	2	7	9	1
Jan. 6, '45	71	0	1	2	24	0
Jan. 13, '45	124	0	1	1	13	0
Jan. 20, '45	258	0	0	1	24	0
Jan. 27, '45	196	0	1	0	6	0
Feb. 3, '45	141	0	1	2	6	0
Feb. 10, '45	104	0	0	3	8	0
Feb. 17, '45	87	0	1	3	8	2
Feb. 24, '45	88	0	1	2	5	0
Mar. 2, '45	94	0	2	3	10	1
Mar. 9, '45	78	2	1	3	14	0
Mar. 16, '45	119	0	3	6	21	1
Mar. 23, '45	153	0	1	1	22	1
Mar. 30, '45	182	1	2	2	12	0
Apr. 6, '45	154	0	1	2	12	1
Apr. 13, '45	153	2	1	3	18	2

same period there were only four rheumatic fever admissions, whereas during a like period immediately prior to prophylaxis there were 10 such admissions and again 10 admissions during the same interval immediately following cessation of the program (table 1). There was a steady diminution in the number of common respiratory admissions during prophylaxis. Admissions for scarlet fever declined only slightly. Lobar pneumonias declined by approximately one fourth. We are unable to explain the sharp drop in admissions for atypical pneumonia. These facts will be commented upon more fully in the discussion.

TABLE II

Hospital Admissions for Untoward Sulfadiazine Reactions

Case	Temperature	Rash	Clinical Findings	Laboratory for Throat Culture and White Blood Cell Count	Admission Diagnosis	Progress
1	98.6	Maculopapular generalized	Asymptomatic except for conjunctivitis	Not done	Sulfadiazine rash	Immediate remission
2	99.2	Scattered maculopapular on face	Post-cervical adenopathy itching	W.b.c. 4000—polys. 57, lymphs. 37, monos. 4	Obvn. German measles	Immediate remission
	Arriving at barracks given 1 gm. of sulfadiazine—within 2 hours began to feel sick and was re-admitted with:					
	102.8	Generalized maculopapular	Malaise	Not done	Sulfadiazine rash	Remission in 24 hours
3	98.6	Maculopapular on back, face, trunk	Edema of wrists and face, pharynx-injected	W.b.c. 8800—polys. 43, lymphs. 40, monos. 9, eosinophiles 8	Sulfadiazine rash	Remission in 12 hours
4	102.6	Maculopapular generalized	Pharynx-injected	W.b.c. 4600, polys. 59, lymphs. 32, eos. 3	Sulfadiazine rash	Remission in 24 hours
5	101.4	Maculopapular on back, trunk, hands and knees	Pharynx-injected, malaise	W.b.c 10,200, polys. 67, lymphs. 21, monos. 7, eos. 5	Sulfadiazine rash	Febrile for 4 days
6	98.6	Faint erythematous flush, generalized	Asymptomatic	Not done	Sulfadiazine rash	Immediate remission
7	102	Confluent erythematous flush over back and trunk and face	Acute rhinitis Pharynx-injected	Not done	Acute nasopharyngitis	Remission in 24 hours
	Back at barracks given 1 gm. sulfadiazine, fainted within an hour and admitted with:					
	102.8	Maculopapular generalized	Malaise	W.b.c. 5100, polys. 53, lymphs. 42, monos. 4	Sulfadiazine rash	Remission in 24 hours

TABLE II—*Continued*

Case	Temperature	Rash	Clinical Findings	Laboratory for Throat Culture and White Blood Cell Count	Admission Diagnosis	Progress
8	102.8	Faint erythematous flush on face and trunk	Malaise	Not done	Sulfadiazine rash	Remission in 12 hours
9	102	Confluent erythematous over trunk	Post-cervical adenopathy headache, pharynx-injected, acute rhinitis	Not done	Acute nasopharyngitis	Immediate remission
			Arriving at barracks took 1 gm. of sulfadiazine—became ill shortly after with:			
	102.8	Generalized scarlatiniform	Malaise, nausea	W.b.c. 6500, polys. 67, lymphs. 25, monos. 5, eos. 2	Sulfadiazine rash	Remission in 24 hours
10	98.6	Maculopapular on both forearms and groins	None	Not done	Sulfadiazine rash	Immediate remission
11	100.2	Scarlatiniform on thorax and arms	Pharynx-injected, headache, chest pain	Throat culture negative for Beta hemolytic streptococci. W.b.c. 6700, polys. 74, lymphs. 18, monos. 5, eos. 3	Obvn. for scarlet fever	Remission in 24 hours
12	100	Scarlatiniform generalized	Pharynx-injected, malaise, strawberry tongue, axillary adenopathy	Throat culture negative for Beta hemolytic streptococci. W.b.c. 8700, polys. 82, lymphs. 11, monos. 1, eos. 2	Obvn. for scarlet fever	Remission in 24 hours
13	102	Scarlatiniform on thorax	Pharynx-injected, headache	Throat culture negative. W.b.c. 4900, polys. 6, lymphs. 32, monos. 32, eos. 3	Obvn. for scarlet fever	Remission in 24 hours
14	98.6	Maculopapular over trunk and arm	Asymptomatic	Not done	Sulfadiazine rash	Immediate remission

TABLE II—*Continued*

Case	Temperature	Rash	Clinical Findings	Laboratory for Throat Culture and White Blood Cell Count	Admission Diagnosis	Progress
15	99.6	Discrete maculopapular over chest	Pharynx-injected	Not done	Sulfadiazine rash	Remission in 24 hours
16	104	Erythematous punctate over trunk	Generalized lymphadenopathy	W.b.c. 9400, polys. 70, lymphs. 22, monos. 6, eos. 2	Sulfadiazine rash	Remission in 48 hours
	Hospitalized for 10 days for acute nasopharyngitis—discharged and returned to barracks: given 1 gm. of sulfadiazine—taken ill within 3 hours with:					
17	102.4	Faint erythematous, generalized	Malaise and pain in bones and joints	Not done	Sulfadiazine rash	Remission in 36 hours
18	98.6	Generalized maculopapular	Asymptomatic except for itching	Not done	Sulfadiazine rash	Immediate remission
19	98.6	Fine maculopapular over body	Asymptomatic	Not done	Sulfadiazine rash	Immediate remission
20	98.6	Maculopapular on face and shoulders	Post-cervical adenitis	Not done	Obvn. for German measles	Immediate remission
21	98.6	Maculopapular on face and chest	Asymptomatic	Not done	Sulfadiazine rash	Immediate remission
22	100.2	Brownish erythematous flush, face, trunk, extremities	Post-cervical adenitis, pharynx red	W.b.c. 6800, polys. 69, lymphs. 21, monos. 7	Sulfadiazine rash	Remission in 36 hours

TABLE II—*Continued*

Case	Temperature	Rash	Clinical Findings	Laboratory for Throat Culture and White Blood Cell Count	Admission Diagnosis	Progress
23	100	Scarlatiniform over chest and back	Asymptomatic	5200, polys. 70, lymphs. 21, monos. 6, eos. 3	Sulfadiazine rash	Remission in 24 hours
24	98.6	Urticaria generalized	Asymptomatic	6400, polys. 64, lymphs. 36	Sulfadiazine rash	Remission in 24 hours
	Became ill after 1st dose within 3 hours					
25	101.6	Erythematous flush over body	Malaise	7200, polys. 62, lymphs. 20, monos. 11, eos. 7	Sulfadiazine rash	Remission in 24 hours
26	101	Maculopapular neck, face and chest	Pharynx-injected, post cervical gland	4500, polys. 55, lymphs. 38, monos. 4, eos. 2	Obvn. German measles	Remission in 24 hours
27	102.4	Scarlatiniform generalized	Pharynx-injected	10,000, polys. 91, lymphs. 3, eos. 1, monos. 5	Obvn. scarlet fever	Remission in 48 hours
28	99	Erythematous flush	Asymptomatic	Not done	Sulfadiazine rash	Immediate remission
29	98.6	Maculopapular trunk and upper extremities	Asymptomatic	Not done	Sulfadiazine rash	Immediate remission
30	98.6	Urticaria over chest and trunk	Asymptomatic	Not done	Sulfadiazine rash	Immediate remission

Reactions to Prophylactic Sulfadiazine. The untoward reactions to prophylactic administration of sulfadiazine were studied as two groups: those admitted to hospital and those observed and treated as out-patients. Table 2 is a résumé of the 30 cases hospitalized. A glance shows that among the patients five (cases 11, 12, 13, 23, and 27) were admitted for observation for scarlet fever, and three (cases 2, 20 and 26) were admitted for "possible German measles." It was only after negative throat cultures for beta hemolytic streptococci and observation of the clinical progress that these diseases were eliminated. The rashes consisted of maculopapular, faint confluent erythematous, and scarlatiniform eruptions. With the exception of case 16 which showed a temperature of 104° F., the temperature ranged between 99.6° and 102.8° F. All patients except cases 16 and 27 (48 hours) and case 22 (36 hours) became afebrile within six to 24 hours after admission. Eleven patients showed injected and diffuse pharyngitis. Post-cervical adenopathy was present in four patients and one had a generalized lymphadenopathy. Two patients (cases 2 and 26) had a white blood cell count of 4000 and 4500 respectively which is below the accepted level of leukocyte count.[7] Under normal conditions from 2 per cent to 4 per cent of leukocytes found in adult human blood are eosinophiles. Three patients, cases 3, 25 and 5, showed an eosinophilia of 8 per cent, 7 per cent, and 5 per cent respectively.

Among the ambulatory cases, there were 33 patients with mild untoward reactions who were seen and treated by the dispensary surgeons. Table 3

TABLE III

Ambulatory Patients with Untoward Sulfadiazine Reactions

Rash (urticaria, maculopapular, scarlatiniform)	Edema (eyelids, cheeks, wrists)	Gastrointestinal (nausea, vomiting)
22	5	6

is a résumé of these cases. These comprise 22 patients with skin reactions, consisting of urticaria, maculopapular and scarlatiniform rashes; five cases of mild angioneurotic edema involving eyelids, cheeks and wrists; and six cases of gastrointestinal complaints consisting of nausea and vomiting.

COMMENT

The following is stated in the Bulletin of the U. S. Army Medical Department[6]: "Chemo-prophylaxis offers a promising means of controlling a group of diseases which always have been a serious threat to military groups, especially recently inducted troops. There is ample reason to believe that it will markedly reduce the morbidity and mortality of these diseases, will diminish interference with training programs on account of sickness and

lessen the incidence of crippling sequelae and complications." Our results with mass sulfadiazine prophylaxis substantiate these statements. At a time when troops are urgently needed for overseas replacement, we have been able to conserve countless man-days necessary for training by cutting the incidence of certain diseases and hospital admissions. During the period of chemoprophylaxis our weekly admission rate for common respiratory disease was reduced by 33 to 59 per cent. It is worthy of comment that we had two meningococcic meningitis admissions within two weeks after cessation of prophylaxis, the subjects being recent inductees who arrived in camp after the program had been concluded. There was a noticeable drop in lobar pneumonias to 11 cases compared with 15 and 17 during like periods before and after prophylaxis, respectively. The drug had slight influence, if any, upon the incidence of scarlet fever (table 1). It would appear, therefore, that sulfadiazine prophylaxis as employed here exerts no appreciable effect on the prevention of this disease. Ratner [8] frowns upon the therapeutic use of sulfonamides in scarlet fever and quotes numerous observers who doubt the efficacy of the drug in lowering the incidence of complications of this disease. Our study reveals a sharp drop (36 per cent) in the number of atypical pneumonias admitted to hospital during the program. It is difficult to comprehend the reason for this drop. Both chemotherapy and serotherapy have been found to have no influence upon virus disease. Conjecture upon the factors underlying the diminished incidence of atypical pneumonia is not within the scope of this paper; nevertheless, it may not be amiss to suggest further investigation along these lines. Our results showed a marked diminution in rheumatic fever episodes during chemoprophylaxis, admissions having been cut by 60 per cent. It must, of course, be borne in mind that all soldiers in this program (except those hospitalized) remained on a full duty status. The duties of an infantry trainee at this Infantry Replacement Training Center are rigorous indeed and certainly conducive to recurrences of rheumatic episodes in diathetic individuals. It may be assumed, therefore, that the incidence of rheumatic fever would have been diminished even farther if we had been able to limit the activities of individuals with rheumatic histories.

Analysis of our untoward reactions from mass sulfadiazine prophylaxis reveals the figure to be very low. There were no serious reactions. Among approximately 20,000 individuals taking a daily one gram dose of sulfadiazine for a period of five weeks there were 63 who reacted adversely. Thirty of these were admitted to the hospital for further study and treatment; the remaining 33 were treated at the dispensaries.

It is interesting to note that among the 30 hospital cases, five had an admission diagnosis of "observation scarlet fever," and three were admitted for "observation German measles." The similarity between the reaction due to sulfadiazine and the symptoms of early scarlet fever and German measles is such that sometimes only after appropriate laboratory tests and clinical progress could the correct diagnosis be established. Therefore, one must ques-

tion patients whether they have recently received sulfonamide drugs when faced with apparent acute exanthematous infections. Skin eruptions were present in 52 of all reactors, while 30 had pyrexia and rash. Sore throat and malaise were common symptoms.

It is not within the scope of this paper to enter into a detailed discussion concerning the mechanism of sulfonamide reaction. There is no doubt that primary toxic reactions do occur after sulfonamide therapy, particularly when given in large amounts. Ratner [8] stresses that up to the present time all aberrant reactions to these drugs have generally been classed together indiscriminately under the designation of "toxic reactions." He points out that as these drugs are used for prophylaxis and therapy repeatedly in a wide variety of diseases untoward reactions will occur which are not toxic reactions but rather the development of hypersensitivity to the drug. A careful study of our cases seems to verify Ratner's hypothesis. One of us (BWB) personally interrogated more than one-half of the reactors.[36] Fourteen among them stated that they had taken at one time or another some form of "sulfa drugs" with impunity. Six others thought they were given sulfa tablets by their physicians for colds, but were not certain; the rest professed ignorance about having taken the drug before. Among the reactors several developed a reaction comparable to serum sickness, i.e., an incubation period of six to 14 days after beginning prophylaxis, an abrupt febrile onset, malaise, rash, itching and nausea and eosinophilia. Others on the other hand reacted immediately upon taking the drug, resembling the so-called accelerated type of serum allergy. The case history of patient 2 may be cited as an example. The patient had been receiving prophylactic sulfadiazine for about 12 days when he developed a scattered maculopapular eruption of the face, itching, and posterior cervical adenopathy. He was afebrile and was admitted with a diagnosis of observation for German measles. He improved rapidly and after four days was discharged. Arriving at his barracks he was given 1 gram of sulfadiazine. Within two hours he began to feel sick, developed generalized maculopapular rash with a temperature of 102.8° F., and was readmitted, this time with the proper diagnosis. Cases 7, 9, 17, 25 and 27 present similar histories. One may further conclude that the repetition of sulfadiazine even in small doses in a previous reactor to the drug may precipitate an immediate untoward reaction.

Summary

1. Mass sulfadiazine prophylaxis was instituted for a period of five weeks after hospital admissions for common respiratory diseases reached an alarmingly high rate with 23 per cent due to hemolytic streptococci clinically.

2. One gram of sulfadiazine was administered daily to about 20,000 soldiers.

3. During chemoprophylaxis our hospital admission for common respiratory diseases dropped by 33 per cent at the end of the first week.

4. There were no cases of meningitis during the period of prophylaxis whereas there were two admissions within the following two week period.

5. There was a marked diminution of episodes of rheumatic fever during sulfadiazine prophylaxis.

6. There was a noticeable drop in lobar pneumonias during the program.

7. There was a drop of about one-third in admissions for atypical pneumonia. We do not know whether this finding was coincidental or related to our program.

8. Sulfadiazine prophylaxis did not appreciably influence the incidence of scarlet fever.

9. There were no serious reactions to the drug. However, among the total of 63 reactions, 30 were admitted to the hospital for further study. Thirty-three were observed and treated as out-patients.

10. Among the 30 hospital cases, five were admitted with a tentative diagnosis of scarlet fever and three of German measles. It was only after negative throat cultures and observation of clinical progress that these diseases were eliminated. All hospitalized patients had fever of short duration and skin eruptions.

11. Among the 33 ambulatory cases, there were 22 patients with skin reactions consisting of urticaria, maculopapular, and scarlatiniform eruptions; five with mild angioneurotic edema; and six cases of gastrointestinal complaints consisting of nausea and vomiting.

12. We feel that at least in some instances reactions analogous to allergic sensitizations occurred, manifested by urticaria, angioneurotic edema, and gastrointestinal symptoms. Ratner's hypothesis that reactions are manifestations of sensitization as apart from toxic phenomena is discussed and evidence is offered to support this view. We offer a classic example in case 2 discussed above. We caution against administration of sulfadiazine even in small doses in the presence of a history of previous reactions.

13. In military establishments where troop population is constantly augmented by men fresh from civilian life, routine administration of sulfadiazine prophylaxis appears to be a potent means of reducing non-effective rates. By diminishing the incidence of certain diseases, thereby reducing time lost from training, chemoprophylaxis makes a significant contribution to the winning of the war. Its similar and timely application in civilian life, especially in institutions such as schools, camps, asylums, etc., is fully as promising in reducing the incidence of certain diseases and their sequelae.

14. We are stimulated to further study of reactions to this drug.

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PEPTIC ULCER IN IDENTICAL TWINS *

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The literature concerning the familial incidence of peptic ulcer was reviewed in this Journal in 1933,[1] and although the reports suggested an inherited factor prominent in the basic causes of peptic ulcer, I was unable at the time to find the disease in identical twins or any reference to it in the literature. Since 1933, three instances have appeared in the literature. I wish to report the fourth instance of the occurrence of this pathological process in identical twins as further evidence in support of the hereditary nature of peptic ulcer.

It is rational, in view of the limitation of our present knowledge, to assume that gastric and duodenal ulcers are fundamentally the same disease. In this paper the term *peptic ulcer* refers to the lesion in either location.

In 1935, E. Schindler [2] reported a study of 39-year-old twin brothers who had ulcers of the lesser curvature of the stomach perforating within one month of each other. The diagnoses were confirmed by operation. The family history of these twins contained instances of both cancer of the stomach and peptic ulcer.

In the same year, F. von Mentzingen [3] reported cases of 20-year-old identical twin sisters with roentgenographic evidence of duodenal ulcer, the father of these twins having had a gastroenterostomy for pyloric obstruction.

In 1944, McHardy and Browne [4] reported duodenal ulcer concomitant in identical male twins, aged 28. This report does not include a family history.

In 1938, C. W. Kidd [5] reported similar symmetrical and simultaneous duodenal ulcers in dizygotic male twins, which perforated within an hour of each other. The diagnoses were confirmed by operation. Since these were not identical the report cannot be used to substantiate our thesis because peptic ulcer in siblings is strikingly common, although to have the accident of perforation occur in each within an hour is an unusual coincidence.

In three of these instances then the peptic ulcers occurred in homologous twins; in the fourth they occurred in dizygotic twins.

The occurrence of cancer of the stomach in these families with peptic ulcer, as mentioned in one of the reports above, is between four and six times as great as in families in which ulcer does not occur, so that it is not surprising that Militzer [6] was able to report in 1935 the occurrence in 70-year-old male twins of symmetrically located cancers of the stomach, with simultaneous, identical symptoms and similar lesions. This instance of cancer of the stomach in twins does not necessarily support the commonly accepted

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theory that gastric ulcers may be precancerous lesions but it does add interest to genetic relationships of the two gastric lesions.

Case Reports

An identical twin (Margaret), aged 20, complained of a gnawing sensation in the epigastrium after meals and awakening her at night. Food gave relief. The symptom usually was more pronounced in the spring with relief during the summer and winter. There had been no vomiting and no tarry stools. The symptoms appeared about four weeks after the birth of her first child, when she was 18 years old.

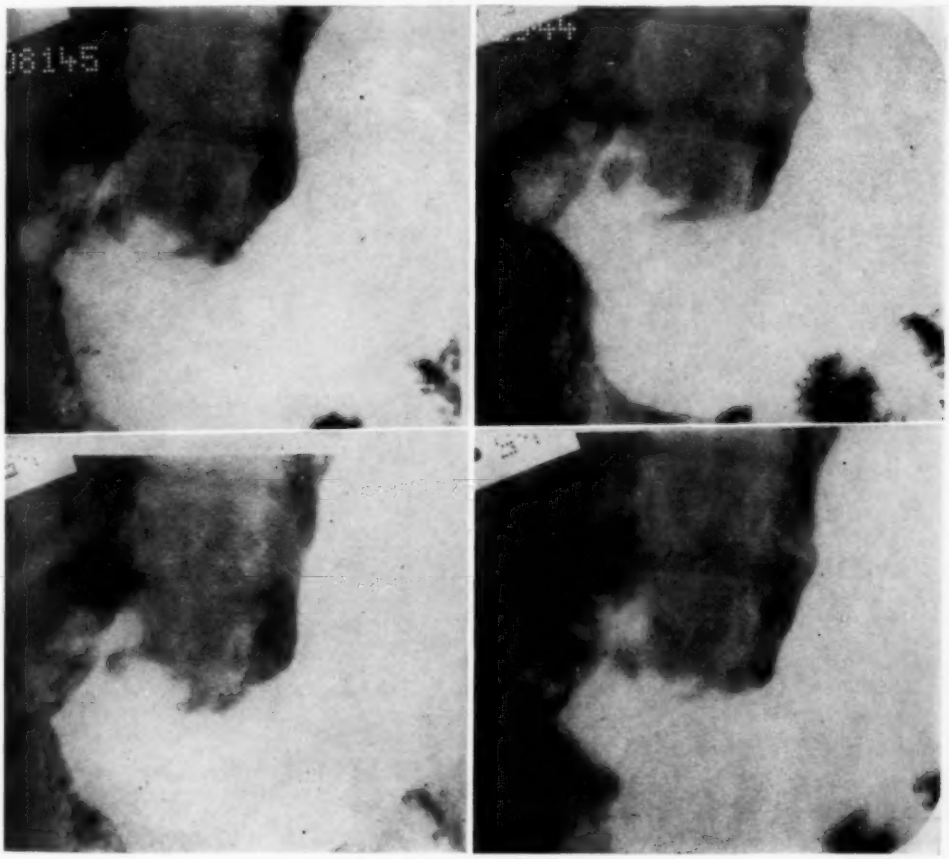

Fig. 1. (Margaret) Showing ragged irregular appearance of the cap in the postero-anterior and lateral projections.

Roentgen studies by Dr. S. M. Donaldson showed an irregular, ragged appearance of the duodenal cap in the posterior-anterior and in the lateral projection (figure 1). A diagnosis of chronic duodenal ulcer was made.

The other twin (Marian) had epigastric distress four months after the birth of her second baby, the first having been stillborn about two years before. Her symptoms were similar to those of Margaret. The roentgenographic studies by Dr. G. T.

Patrick showed extreme tenderness on pressure over the duodenal cap, a marked irregularity of the cap, and a diagnosis of chronic duodenal ulcer was made (figure 2).

The father of these twins had stomach trouble for a number of years and in 1938 a gastric resection for duodenal ulcer was made at the University of Michigan Hospital. The roentgen and clinical evidence in the father's case was unequivocal.

The characterology of these twins follows that consistent in peptic ulcer cases, in that it emphasizes the extreme meticulousness, scrupulousness, and

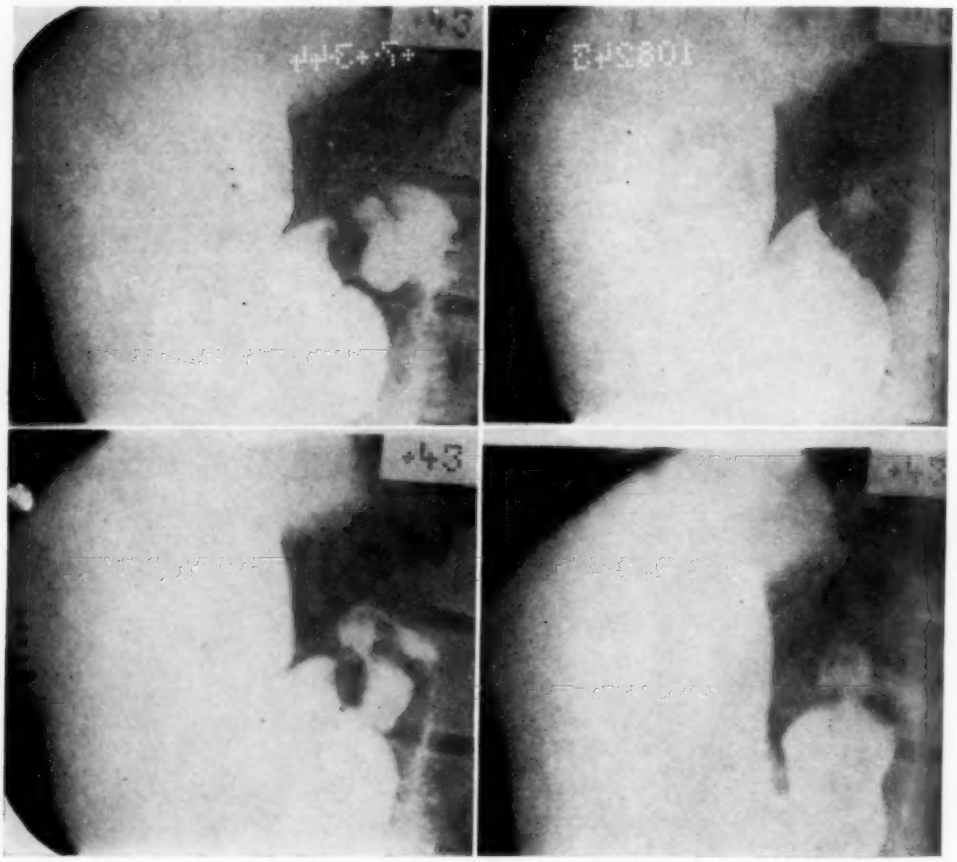

FIG. 2. (Marian) Showing no definite complete filling of the duodenal bulb. The irregularity confirms that noted by the fluoroscope.

neatness of the patients, and their inability to tolerate "disorderliness," all of which is typical of the personality pattern of the ulcer patient. Activation of an ulcer, sometimes with hemorrhage or perforation, occurs when unusual psychic tension referable to these personality characteristics is experienced.

The Identity of Twins. There is always some difficulty in determining whether or not twins are homozygotic. Identity of sexes is not sufficient proof. According to the mother's statement there was only one placenta

at the birth of the twins, Margaret and Marian. In appearance they were identical as far as the eye could determine. They married brothers. They had attacks of acute appendicitis within 11 days of each other. Their actions, expressions, and mannerisms were identical. Margaret weighed one and one-half ounces more than her sister at birth, and now weighed ten pounds more than her sister. Their menstrual histories were identical. Their blood groups were the same. The deliveries of their babies were uncomplicated. Their blood pressures were identical.

We attempted to substantiate further the identity by fingerprints and electrocardiograms. The fingerprints were referred to Lieutenant H. E. Ericson, of the Michigan State Bureau of Identification of the Michigan State Police Department, with the following note:

"The fingerprint classification of these prints indicates that there are no similarities between these prints of twins. There is nothing unusual about either set of prints. Numerous sets of prints of twins have been received in the past and occasionally there are similar characteristics but in this case there is nothing which might indicate that they were fingerprints of twins. The following is the fingerprint classification in each case.

Margaret	(19)	L	9	R	O	18
		S	1	Ua	aO	10
Marian	(18)	L	1	A	OO	5
		M	1	U	OI	14"

Since the literature commonly states that identical twins have either identical or "mirror image" fingerprints, this note should be of some interest.

It has been suggested that the electrocardiograms are identical in twins, but Wise, Comeau, and White,[7] among others, found that electrocardiograms are not necessarily similar in identical twins. Electroencephalograms and Rorsach tests were not obtained on these subjects, nor was the phenylthiocarbamide test used.

In the case of Margaret and Marian, the evidence of identity will have to rest upon the similarity of their physical and mental characteristics and the statement of the mother that there was a single placenta at birth.

Discussion

The observation of the occurrence of ulcer in identical twins introduces several questions. One must agree that the tendency to ulcer is inherited. The mechanism will be understood only after a complete genetic study of all the members of several generations. Do not these patients really inherit a personality type which activates predisposed but unknown components of the autonomic nervous system thus producing hyperchlorhydria or ulcer or both when under emotional tension? A similar situation certainly exists in many other hereditary diseases in that of all the detectable factors making up an inherited clinical entity, some members of the family may show only one or two. We may assume then, that the three most constant factors constituting the clinical entity of peptic ulcer may not appear in all members of a given ulcer family. It is, of course, possible that the hyperchlorhydria accompanying duodenal ulcer may be familial, may be incidental or secon-

dary, and may or may not be related to personality types. It is also possible that separate genes are required for the inheritance of hyperchlorhydria and the personality type, and that the combination in the same person would give rise to the susceptibility to ulcer.

There can be no doubt that peptic ulcer is a familial disease. The evidence for a hereditary component is overwhelming. Both prevention and treatment, if successful, will depend upon a correct evaluation of an inherited personality pattern upon which psychic trauma causes ulcer formation by mediation through known reflex arcs between cerebrum, hypothalamus, and vagus nerve, and the gastric and duodenal mucosa.

It is obvious that the *basic* cause of peptic ulcer is not related to environmental, hormonal, or nutritional factors, although the cholinergic components may be secondarily involved. In this respect interest is being shown in the appearance of coronary thrombosis and peptic ulcer in the same person. Three such instances accurately diagnosed have come to my attention and in one the autopsy showed both lesions to be recent. Coronary thrombosis also occurs in families and reports of the disease are likely to appear concerning identical twins. Both diseases have strong emotional components, both are produced experimentally by vagal stimulation and in animals dying of adrenal insufficiency.

It will be necessary to leave the final answer to these questions to the development of experimental medicine and psychiatry.

Summary

Cases of duodenal ulcer in identical female twins are reported. This is the fourth instance of its kind reported in the current literature and further confirms the basically hereditary nature of peptic ulcer.

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DIPHTHERIA CARRIERS TREATED WITH PENICILLIN *

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The majority of patients with diphtheria rid themselves of bacilli within the first two weeks after recovery. However, there remains a fairly large percentage of cases who become carriers and who present a serious danger to the spread of the disease unless isolated or freed of the infecting organism. Heretofore, although a great variety of bactericidal agents has been tried, the treatment of carriers has remained unsatisfactory.

It has previously been shown by Abraham, Chain, et al.[1] Chain, Florey et al.,[2] and by Fleming,[3] that *Corynebacterium diphtheriae* is susceptible to the action of penicillin in vitro. Because of this fact, and because of the large number of diphtheria carriers encountered on our service in one of the army general hospitals, Colonel William S. Middleton, Chief Consultant in Medicine for the ETOUSA suggested that we try the use of penicillin to solve this problem.

Experimental Data

1. Laboratory Studies. The carrier state was judged by the results of examination of smears taken from cultures grown on tellurite medium. Swabs from the throat and from the nasal mucous membranes were cultured on selected potassium tellurite media, and typical black colonies were fished and confirmed microscopically by stained films. Cultures on tellurite media frequently yield short bacilli with typical granules. Any questionable cases were subcultured on Loeffler's media which yields a more typical organism for microscopic study. Albert's staining technic was used in the diagnostic slide procedure.

As pointed out by Helen A. Wright,[4] positive results from throat cultures may be misleading because "the biological distinction between *Corynebacterium diphtheriae* and related non-pathogenic species is not always sharp." Due to lack of facilities at this hospital, virulence studies were not carried out.

Five consecutive negative nose and throat cultures served as our criteria for release from isolation.

2. Systemic Treatment. Nine patients who had recovered from clinical diphtheria and continued to show positive throat cultures for from two to seven weeks after symptoms had subsided were given intramuscular injections of 25,000 units of penicillin every two hours for seven days, for a

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total of 2,100,000 units. As may be seen from table 1 (first five cases) and table 2 (four cases), cultures remained positive in all nine cases treated.

3. Local Treatment with Penicillin. The failure of systemic treatment with penicillin becomes understandable when we stop to consider the nature of the diphtheria bacillus. It is well known that this organism has but slight capacity to invade the body and it is found almost exclusively on the surface

TABLE I

Penicillin Therapy for Diphtheria Bacillus Carriers
Systemic and Local Treatment

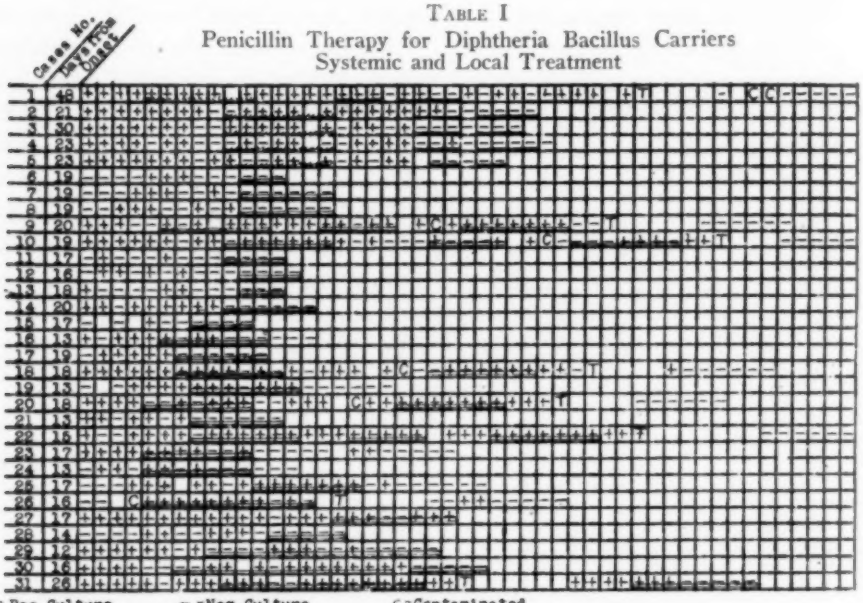
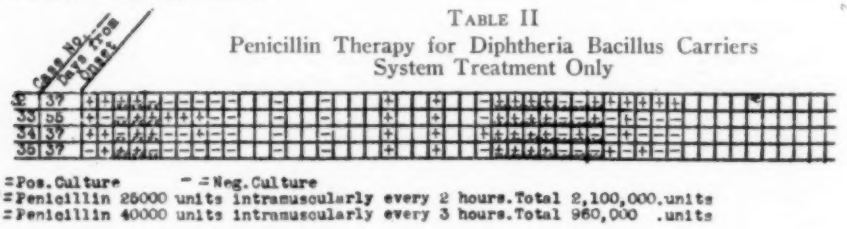


TABLE II

Penicillin Therapy for Diphtheria Bacillus Carriers
System Treatment Only



of the affected mucous membranes. The symptoms and harm produced by the disease are due entirely to the soluble toxin absorbed from the surface of the membranes affected. It would, therefore, seem logical to try the effect of topical application of the bacteriostatic agent. However, penicillin, to be effective, requires more than momentary contact, such as would be produced by painting the surface or by the use of infrequent sprays. In a personal communication from Professor Sir Alexander Fleming, the use of mouth lozenges was suggested. With a little experimentation, our pharma-

cist devised a glycerine and gelatin lozenge that required 20 to 30 minutes to dissolve, when held between the cheek and gums, thus giving prolonged contact with the throat by the released penicillin (formula given below).

Base Presently Used for Penicillin Lozenge:

Agar	30.0 Gm.
Water	500.0 c.c.
Glycerite of Starch	350.0 Gm.
Sucrose	720.0 Gm.
Penicillin	400,000 units
To make	400 Lozenges.

Alternate Base:
Glycerinated Gelatin

Gelatin	800.0 Gm.
Glycerin	640.0 c.c.
Water	q.s. to wet Gelatin
Penicillin	400,000 units
To make	400 Lozenges.

A total of 31 diphtheria carriers was treated with penicillin lozenges. Each patient received one lozenge every hour for 12 doses during waking hours over a period ranging from three to 15 days. The first patients treated received 500 units per lozenge; after January 1, 1000 units per lozenge was used. Because of the possibility that the nasal mucous membranes also might harbor bacilli, penicillin sprays containing 1000 units per c.c. were given, every two hours, during the same period. Results are shown in table 1.

Discussion of Results

Of the 31 diphtheria carriers treated with the lozenges and penicillin nasal spray, 23 or 74 per cent cleared promptly, the cultures becoming consistently negative from the first to the tenth day after beginning treatment. The eight cases in this series that remained positive despite treatment were found to have enlarged cryptic tonsils. We then had these eight patients' tonsils removed, following which seven gave consistently negative cultures without further treatment. When penicillin lozenges and nasal spray treatment were then applied to this one exception over a period of eight days, the cultures from the nose and throat became free from the infecting organisms.

Case 30 was treated at first with the lozenges alone, for a period of 13 days, without success in clearing both the throat and nasal secretions; however, when nasal spray was added to this treatment, they cleared promptly.

The fact that cultures became negative only after tonsillectomy in seven of the cases is considered proof that the tonsils are important sites for harboring diphtheria bacilli. That the nasal passages also may serve as the focus was evidenced by two cases. In one case (No. 31), nasal cultures remained positive after tonsillectomy. In the other (case No. 30), cultures became negative only after nasal spray treatment was added to lozenges.

SUMMARY

1. Treatment of diphtheria carriers by intramuscular injections of penicillin in adequate dosage over a period of seven days was found to be ineffectual in ridding patients of the diphtheria bacillus.

2. Penicillin lozenges containing 500 and 1000 units per lozenge, taken by mouth, in combination with a nasal spray containing 1000 units per c.c., resulted in five consecutive daily negative cultures in 23 out of 31, or 74 per cent of carriers treated.

3. Eight cases who failed to clear with this treatment alone promptly became negative after tonsillectomy without further penicillin treatment, with one exception. This case cleared readily when again treated with the lozenges and spray.

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HYPOGLYCEMIA IN NEUROPSYCHIATRY *

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Introduction

The neuropsychiatric manifestations of hypoglycemia have been described in the literature. Duncan [1] reported the case of a 19-year old girl who complained of attacks of vertigo and headaches. There had been spells of weakness and one morning she became emotionally upset and lost consciousness. A pancreatic adenoma was removed at operation, this having been a case of organic hyperinsulinism.

Conn,[2] in his excellent monograph on the spontaneous hypoglycemias, reported a case of multiple pancreatic islet cell adenomata, the manifestations having been an "aura" of epigastric discomfort and numbness of the lips and the extremities, followed by a peculiar, fixed, facetious facial expression; change in personality; diplopia; mental confusion; and disorientation associated with jerking movements of all extremities and jaw muscles, and then generalized convulsions.

Helfer [3] reported a case in which the manifestations of hypoglycemia were dizzy spells, blindness, headaches, fatigue, and periods of amnesia.

Rayner and his associates [4] reported a case of hypoglycemia in a 46-year old woman. During one spell of unconsciousness the patient was incontinent of urine and about the same time neurologic signs, patellar and ankle clonus and plantar responses, were elicited. A diagnosis of hysterical fits had previously been made.

Conn [2] reported the case of a 16-year old girl who had periodic attacks of unconsciousness occurring once or twice a month, each attack usually beginning with visual disturbances, followed by involuntary shaking of the extremities and irrational chattering. Following the attacks, a mild disorientation persisted. A diagnosis of epilepsy had been made, but studies revealed this to be a case of functional hyperinsulinism resulting in hypoglycemia.

Another of Conn's patients,[2] a 47-year old man, suffered from attacks characterized by excessive perspiration, vomiting, incontinence, drowsiness, disorientation, followed by unconsciousness. The attacks ended gradually, but the patient remained disoriented for many hours and there was complete amnesia for the attacks. A diagnosis of intracranial tumor had been made. The hypoglycemia in this patient was discovered to be hepatogenic.

Ziegler [5] reported the case of a middle-aged man who was troubled by somnambulism, states of confusion, and amnesia. After hospitalization for

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Since this article was submitted for publication, the author has resumed his civilian practice in Detroit, Mich.

psychiatric observation he was at times maniacal. It was discovered that his symptoms occurred concomitantly with a dangerously low blood sugar level.

McClenahan and Norris [6] reported the case of a truck driver, previously very careful and never arrested, who began to have attacks of amnesia during which he committed numerous traffic violations. These attacks were found to be due to hypoglycemia.

The neuropsychiatric manifestations of insulin reactions in diabetic patients have been the subject of numerous reports in the literature. Disorientation, emotional instability, difficulty in concentration, mental confusion, faintness, diplopia, and cardiac palpitation have occurred separately or in combination. Headaches are frequently encountered, especially with the slowly acting protamine zinc insulin. Manifestations are at times mistaken for those due to alcoholic intoxication, especially combinations including dizziness, impairment in gait, and slurred speech. As an example of the emotional and personality changes of this type of hypoglycemia the case reported by Leyton is cited. A diabetic "invited some friends to a meal and began by pressing them to help themselves more liberally to pepper; then, most excitedly in a loud voice, he hurled insulting epithets at his wife who, recognizing the condition, besought him to take some sugar. He replied that of course she wanted him to take sugar, a thing the doctor had forbidden him, so that she might get rid of him, and marry someone else. This patient was finally forced to take sugar; the symptoms passed within half an hour and he had no recollection of what occurred." [7]

So bizarre a pattern of signs and symptoms may occur in the presence of hypoglycemia, and so frequently emotional and psychic disturbances, that a variety of erroneous diagnoses have been made. Among these are epilepsy, narcolepsy, intracranial tumor, diffuse disease of the brain and spinal cord, psychosis, psychoneurosis, neurasthenia, and gastrointestinal disorders.

This close relationship between the signs and symptoms of hypoglycemia and the nervous system represents the effects of low blood sugar concentration on the central and sympathetic nervous system. The extreme sensitivity of the brain to hypoglycemia is due to the fact that it is the only organ which obtains its energy from the combustion of carbohydrate alone. Other organs of the body support their metabolism by oxidation of both carbohydrate and fat. When the blood supply of carbohydrate is decreased, the various non-nervous tissues of the body maintain their activities normally at the expense of energy derived from the oxidation of fat. The brain cannot do this, for it has no foodstuff as an alternate for carbohydrate; its metabolism slows down and the cerebral function suffers.[8]

Furthermore, the newest portion of the brain, the cerebral cortex, has the highest rate of metabolism of all nervous tissue and is therefore the first to suffer from decrease in blood sugar concentration.[8]

Therefore, the possibility of hypoglycemia, whatever the cause be, must be borne in mind, especially in neuropsychiatric work.

Present Data

We recently had occasion to study two patients on the neuropsychiatric service of this hospital in whom this possibility had to be entertained in the differential diagnosis.

Case Reports

Our first patient was a 21-year old private, sent to the hospital with the diagnosis of post-traumatic psychosis. Following an auto accident, seven years before, he had experienced spells of nervousness, jumpiness, and tenseness at loud noises; he had had infrequent attacks of headache and blurred vision following which he had become unconscious. In his voyage to this theatre he had fallen during such an attack and had struck his head.

Early in his hospital stay he was observed in several mild attacks; they were preceded by a sensation of "light headedness." He complained of headache and became confused; there were a few light convulsive muscle spasms in the arms and legs; and his speech was incoherent and barely discernible. In one attack he wandered to the wrong ward at bedtime and prepared to retire; it was necessary to help him back to his ward because of difficulty in his gait. In another attack he fell to the floor unconscious, and had a mild convulsive seizure. There was a total amnesia for the attack.

After preparation on a diet yielding at least 355 grams of available glucose daily,[9] a glucose tolerance test revealed these values:

Fasting	80 mg. per 100 c.c.
1st hour	100 mg.
2nd hour	95 mg.
3rd hour	71 mg.
4th hour	91 mg.
5th hour	74 mg.

A glucose tolerance test was then planned, to be performed after a provocative, low-carbohydrate diet,[2, 9] but just prior to the institution of the diet the patient had a moderately severe attack, occurring six hours after his last meal. A blood sugar determination during this attack revealed a concentration of 91 mg. per cent. This ruled out hypoglycemia as the cause of his symptoms.

Our second patient was a 26-year old Private 1st class, sent to the hospital with a diagnosis of chronic syncope. For the past three years, while in the service, he had experienced attacks during which he had "black-out" spells, namely, partial to total loss of vision associated with loss of consciousness, and followed by intense headaches, and then dizziness. They varied in duration from a few minutes to two hours. There was no amnesia.

He had experienced similar attacks for four years prior to his entering the service and during one of these attacks in civilian life he had fallen into a hot aluminum solution and had incurred burns about the face. Some of the attacks occurred prior to meal times, when, he remembered, he had been hungry; some had occurred during the night; however, some attacks had occurred not long after a meal.

Because of the amblyopia, he was studied first on the ophthalmologic service, but no organic ophthalmologic disease was present to account for his symptoms. The patient was accepted on the neuropsychiatric service for further observation.

In the study of his carbohydrate metabolism, he was given first a diet of 310 grams of carbohydrate, 70 grams of protein, and 100 grams of fat, which yielded 355 grams of available glucose daily. Following this preparatory diet,[9] a glucose tolerance test revealed these values:

Fasting	63 mg. per 100 c.c.
1st hour	80 mg.
2nd hour	71 mg.
3rd hour	71 mg.
4th hour	67 mg.
5th hour	74 mg.

The fasting level is relatively low, but not below the critical level of 50 mg. per cent.[2] Though the curve is "flat," there are no hypoglycemic levels, and the curve is interpreted as not abnormal.

The provocative, low carbohydrate diet[2, 9] was then given him. It comprised 25 grams of carbohydrate, 35 grams of protein, and 70 grams of fat, yielding 50 grams of available glucose daily. After three days on this diet, a glucose tolerance test revealed these values:

Fasting	62 mg. per 100 c.c.
1 hour	154 mg.
2 hours	80 mg.
3 hours	43 mg.
3½ hours	60 mg.
4 hours	65 mg.
4½ hours	111 mg.
5 hours	115 mg.

Again the fasting level is low, but not below the critical level. The drop to the hypoglycemic level of 43 mg. per 100 c.c. was accompanied by these clinical findings: The patient began to tremble, was flushed, and began to perspire profusely. He was nervous and "shaky"; had a dull temporal headache, was dizzy, and had diplopia. He stated that he felt just like he had felt in the early parts of his previous attacks—that he was going to faint—but he did not lose consciousness.

This established the diagnosis of hypoglycemia as the cause of his symptoms, and with the data at hand, the basic diagnosis, functional hyperinsulinism, was made.

Discussion

The question arises as to why this patient, who had previously experienced these attacks regularly and frequently, did not have a single spontaneous attack while in the hospital. The explanation seems to be that garrison duty, just as moderate work of any kind, imposed moderate energy demands upon the body metabolism. Furthermore, while in combat, ingestion of food was at times irregular in both time and content. On one occasion in combat, the patient was "pinned down" without food for 36 hours. A further important factor is the release of epinephrine during the entire combat period, reflecting the emotional state, and resulting in fluctuations of blood sugar concentrations.

In contrast to this, while in the hospital, he had access to regular, well-balanced feedings, feedings relatively high in protein content and lower in carbohydrate content than field rations. Furthermore, while on the ward, and later on light duty at the hospital, his activities required but mild exertion. Thus, he was physiologically protected.

Another question arises as to why the first glucose tolerance test did not disclose this abnormality, whereas the second one did. Again, the data at hand provide an adequate explanation. The first test was performed after a full, adequate diet and hence the patient's tolerance was good; that is, he

was able to cope so satisfactorily with a large dose of glucose that no high concentration of glucose occurred post-prandially to stimulate insulogenesis. The effect of the starvation diet, however, was to lower his tolerance. He could not tolerate this high dosage without the building up of a high concentration of sugar in the blood. The insulogenic stimulus level was reached and passed, an excess amount of insulin poured into the blood, and hypoglycemia resulted.

The basic cause of functional hyperinsulinism appears to be an increased responsiveness of the pancreas to the normal insulogenic stimulus, resulting in an outpouring of insulin in amounts greater than required.[2] The treatment then would seem to be along lines aimed at eliminating peaks of post-prandial hyperglycemia. A diet low in carbohydrate and high in protein would seem therefore to be indicated, for the derivation of glucose at a slow, even rate from protein, which in itself is slowly absorbed, would tend to prevent the post-prandial rise in blood sugar.[10, 11] The lower blood sugar levels after meals would really act as insulogenic depressors. This was found to be so [10, 11] and such a diet seems to be the best treatment.

Conclusion

1. Manifestations of hypoglycemia have been presented.
2. Several patients, reported in the literature, have been described.
3. The extreme sensitivity of the central nervous system to hypoglycemia has been pointed out.
4. A patient under neuropsychiatric observation at this hospital, and in whom hypoglycemia was a possibility, has been presented. Adequate study ruled out this diagnosis.
5. Another patient studied on the neuropsychiatric service of this hospital has been reported and along with this a description of the method of study. The suspicion of hypoglycemia as the cause of his symptoms was verified, and the basic diagnosis of functional hyperinsulinism was made.
6. The cause of this disturbance and its dietary treatment have been discussed.
7. And finally, emphasis has been put on the importance of keeping hypoglycemia in mind as a cause of bizarre groups of symptoms, symptoms of so-called "nervous" origin.

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BLOOD PLASMA PROTEINS IN PATIENTS WITH HEART FAILURE *

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THE recognition of oligoproteinemia and more specifically hypoalbuminemia and successes in correction of the significant abnormalities are now commonplace in modern scientific medical practice. Blood plasma protein determination has become practically a routine procedure in diagnostic studies in medical clinics and in our cardiovascular service.

Whipple [1] has summarized the data on the production and utilization, interrelations and modern concepts of plasma protein metabolism. He pointed out that food proteins, after digestion, yielded amino acids which were absorbed from the intestinal tract and carried to the liver. The liver is strategically situated and of sufficient size to perform most of the work of protein synthesis. Some other body cells may perform the function, but most of the plasma proteins seem to emerge from the liver cells and can be utilized in the body for all or most of the protein requirements.

Liver cells can store protein or release fabricated protein. The reserve of plasma protein forming material is considerable, one to five times the circulating mass. The reserve can be reduced by fasting, low protein diet, poor absorption or inadequate rebuilding of plasma proteins or depletion by blood loss. Body protein stores, plasma protein levels, protein production and protein wear and tear are in a state of dynamic equilibrium.

Blood plasma proteins seemingly can pass readily, according to Whipple,[1] from plasma into cells and the reverse without the loss of nitrogen. The proteins need not be reduced to amino acid constituents as formerly thought, but seem to penetrate by preliminary absorption and ultimate penetration into cells. The whole protein on the cell membrane is considered to be modified by contained ferments. The membrane is thought to be composed of bimolecular layer of lipoid molecules between two layers of protein molecules.

Protein on its way out or into the cell is designated as transition protein, which by cleavage and reassemblage will be on its way either to become cell protein or to become plasma protein. Once a cell protein can not be removed it is called indispensable. Any parenchymal cell or tissue can act to store, to utilize, to release and perhaps to fabricate plasma proteins in a small way.

OUR STUDIES

During the past 15 years [2a, b, c, d] we have determined in the clinical biochemical laboratory, rather routinely, the serum proteins and the albumin

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fraction in patients with various diseases, but particularly in those with congestive heart failure with and without hepatic engorgement and edema.

It has been demonstrated that hypoalbuminemia may be the result of one or more of the generally accepted causes. A dilution factor incident to the variable grade but usual hydremic plethora of heart failure must be recognized. The loss of excessive amounts of serum albumin through a defective glomerular filter is admittedly of some significance in patients with congested kidneys, but is more significant in patients with the nephrotic syndrome. There may be lack of building up of the serum albumin or anabolism may be adversely affected as are other hepatic functions in an engorged liver of heart failure. Chavez, Sepulveda and Ortega [3] have demonstrated disturbed liver function tests in patients with congestion of the liver in right ventricular failure. Serum albumin is most markedly

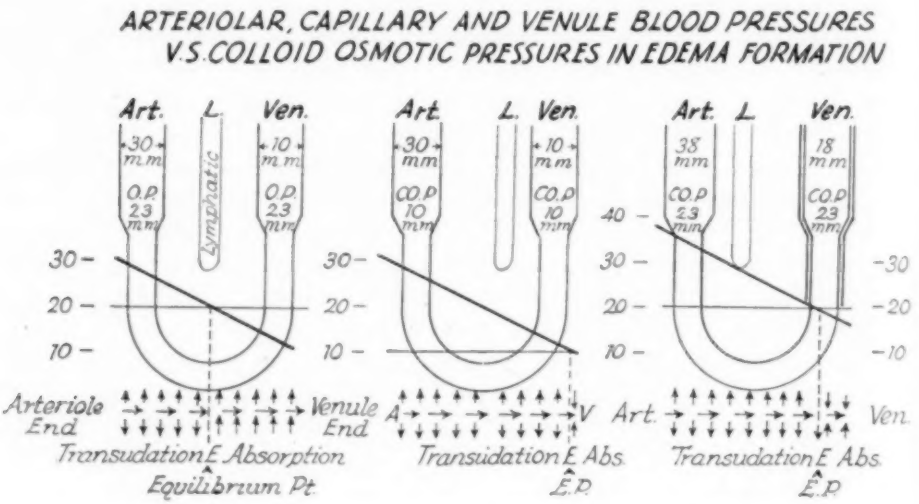

FIG. 1.

decreased when there is disease of the liver, as cirrhosis which may at times be the result of chronic congestion.

Apparently the factor of the gradual development of liver insufficiency that comes with cardiac cirrhosis is important. Katzin, Waller and Blumgart [4] and Garvin [5] have shown that increased fibrous tissue occurs in about one-third of all cases of chronic congestive failure three times as frequently as in all patients that died without congestive heart failure. It, therefore, seems worthwhile to consider liver changes as significant.

A diseased or congested gastrointestinal canal may interfere somewhat with absorption or with the breaking down of complex protein into amino acids for rebuilding. Anorexia may contribute further to decreased intake of food protein of good biological character. Starvation is not as important

a cause of hypoalbuminemia in this country as it is in the enemy occupied and recently liberated countries.

The increased permeability of the capillary walls as the result of toxins or anoxia in patients with severe heart disease might account for edema in some instances. This factor, however, has been practically ruled out by subsequent studies. The rôle of hypoalbuminemia as schematically represented in figure 1, has been minimized in the mechanism of edema of myocardial insufficiency. The postural rise in the venous pressure incident to circulatory failure, intensified by increased hydrostatic pressure in the veins of the dependent part, may be chiefly responsible for edema. The retention of sodium in the tissues as the result of disturbed electrolyte distribution must be considered, though it rarely is the sole factor in edema formation. All three factors combined in various grades usually may be incriminated in the production of edema in patients with congestive heart failure.

It was realized from the beginning that there were several factors concerned in the disturbed water-balance in congestive circulatory failure with edema formation. Venous pressure and the electrolyte levels, as well as the blood serum proteins, were studied in the presence of, during and after the dissipation of the edema.

Standard Methods Used

The total blood proteins have been determined and the albumin fraction and the globulin fraction have been separated by the salting out method and determined by the colorimetric reaction of Folin or by the micro-Kjeldahl and Nesslerization for total nitrogen and subtraction of the non-protein nitrogen. The blood specific gravity, as determined by the falling drop is more simple and probably accurate enough, but is applicable only for total plasma protein levels. We only recently have begun to use it. The absolute total blood volumes and shifts were followed in many cases by application as the new accurate method of Gregerson, Gibson and Stead as modified by Gibson and Evelyn using the Evans Blue Dye No. 1824 and a photoelectric colorimeter. These methods were used in the analysis of blood serum obtained from practically every patient on admission, the morning after diuresis was completed, at weekly intervals, and upon discharge from the hospital.

Present Data

The mass of clinical and laboratory data that has accumulated in our records from the beginning is made up of thousands of determinations and could not possibly be entirely analyzed at this time. Case records of 100 completely studied patients in congestive heart failure were drawn from the files at random. Many of these patients had had numerous studies during various admissions. The great majority of them suffered from hypertensive heart disease, coronary arteriosclerosis and chronic myocardial

insufficiency with congestion of the viscera and edema. There were some cases of syphilitic heart disease and an occasional case of rheumatic heart disease. Most of the patients having frank cirrhosis of the liver were removed from the 100 and substitutions were made.

For comparison the data on 100 patients with edema and congestive heart failure and 46 in whom the edema had cleared are presented in table 1. The normal values that we have obtained previously and those in the literature give mean values of 4.80 gm. per cent (range 4.20 to 5.65) for albumin; 1.90 gm. per cent (range 1.32 to 2.91) for globulin; and 6.70 gm. per cent (range 5.60 to 7.65) for total proteins.

The mean levels for albumin in cardiac patients with edema was 3.54 gm. per cent with a standard deviation of ± .793: for globulin 2.58 gm. per cent with a standard deviation of ± .720 and for total proteins 6.06 gm. per cent with a standard deviation of ± .874. These are slightly but significantly lower than corresponding values obtained later in the same patients.

TABLE I

Blood Serum Proteins in Congestive Heart Failure with and without Edema

	Mean Alb.	Stand. Dev.	Mean Glob.	Stand. Dev.	Mean Total	Stand. Dev.	Mean V.P. cm.	Stand. Dev.
100 Pts. Edema Present	3.54	.793+−	2.58	.720+−	6.06	Grams% .874+−	17.9 (54)	.477+−
Free of Edema (46)	3.62	.667+−	2.65	.797+−	6.12	Grams% .984+−	14.03 (9)	.485+−
Normal Range	4.80	4.20 to 5.65	1.90	1.32 to 2.91	6.70	5.60 to 7.65	9.0	8.0 to 10.0

In 46 patients that became edema-free the mean levels for the albumin were 3.62 gm. per cent with a standard deviation of ± .667; for globulin 2.65 gm. per cent with standard deviation of ± .797; for total proteins 6.12 gm. per cent and standard deviation of ± .984. The differences are relatively small suggesting that full recovery lags behind diuresis.

The venous pressures, on the other hand, in 54 cases during the period of edema showed a mean of 17.9 cm. of water and a standard deviation of ± .477. In the edema-free cases, only nine of which were recorded, the venous pressure was lower with a mean of 14.03 cm. of water and a standard deviation of ± .485, which is a significant difference. The shifts in the electrolytes, particularly in the sodium ion, were not studied in enough cases thus far to warrant giving this element the position of significance that it probably deserves.

For comparison similar data on 20 cases of apparently irreversible cirrhosis of the liver showed lower values; mean albumin 2.58 gm. per cent with standard deviation of ± .536; mean globulin 3.07 gm. per cent with

standard deviation of ± .937; mean total proteins 5.69 gm. per cent with standard deviation ± 1.034 and the mean venous pressure was 13.06 cm. with standard deviation of ± 3.11.

In eight patients with no ascites the mean albumin level was 3.48 gm. per cent with standard deviation of ± .501; mean globulin 2.77 gm. per cent with standard deviation of ± .829; mean total proteins 6.37 gm. per cent with standard deviation ± .706 and mean venous pressure was 11.08 cm. with standard deviation of ± .0.92.

Discussion

Our early studies [2a] (1930),[2b] (1931),[2c] (1932) and those of others [6, 7, 8] suggested that lowered oncotic pressure incident to hypoalbuminemia was at least a recognizable factor in edema formation of congestive heart failure. Stewart [9] disagreed. However, our conceptions are supported by data furnished by further studies which have been subsequently carried out and have not been included in this paper.

Table II

Blood Serum Proteins in Cirrhosis of the Liver with and without Ascites

	Mean Alb.	Stand. Dev.	Mean Glob.	Stand. Dev.	Mean Total	Stand. Dev.	Mean V.P. cm.	Stand. Dev.
20 Pts. Ascites present (20)	2.58	.536+−	3.07	.937+−	5.69	Grams% 1.034+−	13.06 (6)	3.11+−
Free of Ascites (8)	3.48 .501+−		2.77	.829+−	6.37	Grams% .706+−	11.08 (2)	.92+−

The blood serum proteins had been determined in our cardiovascular service in hundreds of other patients with congestive heart failure with edema and after the edema had been rapidly removed by powerful diuretics. Only slight, if any primary effective or persistent plasma protein changes have been recorded during active diuresis in our laboratory.[10] In acute studies with Calvin and Decherd [10] during which great shifts in blood volume were induced in congested cardiacs by diuretics, the albumin fraction was found to shift temporarily as required for maintenance of colloid osmotic pressure. There was evidence that there was an outflow of proteins from and a backflow into the tissues during and after acute diuresis.

The absolute total blood plasma volume has been recognized as increased in congestive failure from 20 per cent to as much as 50 per cent with a slightly greater proportion in erythrocytes than in plasma. However, the decrease on treatment was from 2 per cent to 40 per cent averaging 17.5 per cent with wide fluctuations in many cases.[11]

The delay in the rise in blood plasma proteins after the edema was dissipated and clinical improvement was evident and the reversal of the A/G

ratio and its return to normal support the contention that abnormalities in blood proteins are associated with functional changes in the liver incident to the passive congestion. Apparently the elaboration of albumin follows the slower reëstablishment of the more normal anabolic capacity of the liver cells. The factor of the gradual development of liver insufficiency and hypoalbuminemia that comes with chronic passive congestion and its resulting cardiac cirrhosis has been neglected.

The lowering of the blood serum proteins, particularly the albumin fraction, as a significant effect of hepatic cirrhosis is emphasized in similar data from patients with apparently irreversible cirrhosis of the liver with and without ascites (see table). The patients were generally middle-aged and elderly, about two caucasians to one negro, and two males to one female. In these cases steps had been taken with some success to increase the generally low or depleted blood serum protein mass that had developed as a result of the disturbed metabolic processes in the liver. High protein diets supplemented with yeast, casein hydrolysates, amino acid mixtures or choline and methionine have produced spectacular results in some patients with enlarged fatty livers and hypoalbuminemia, in our experience [12] as well as in the hands of others.

Summary

The rôle of low blood plasma proteins, particularly low serum albumin, in relation to edema formation in patients with congestive heart failure has been investigated.

The results of our previous studies have been substantiated and the blood plasma protein studies on 100 edematous patients with congestive heart failure analyzed. These showed slightly, but definitely subnormal albumin levels with slight compensatory increases in globulin values.

After the dissipation of the edema the blood proteins did not immediately rise to normal levels but there were gradual accretions. It is suggested that the lag may well be due to and evidence of liver dysfunction. Time is required after diuresis and reëstablishment of circulatory equilibrium for liver function to be restored and normal protein anabolism to become effective.

The lowest blood protein levels were noted in patients who had had congestive failure for many months and especially in those who developed evidences of cirrhosis of the liver. Irreversible cirrhosis is shown to result in still lower blood serum albumin values.

Feedings of high protein, acid or neutral ash as well as sodium free, diets are indicated in most patients with congestive heart failure and edema. Proteins of good biological character may be supplemented with protein hydrolysates, amino acids, yeast or choline.

I am indebted to my daughter, Gretchen S. Herrmann, A.B., for stimulating me to work up the material on the first 50 clinical cases that she had analyzed and to Mr. Howard Monk for helping me to complete the study of the other 50 cases and 20 cases of cirrhosis of the liver.

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CASE REPORTS

MASSIVE DOSES OF PENICILLIN IN THE TREATMENT OF SUBACUTE BACTERIAL ENDOCARDITIS*

By Noyes L. Avery, Jr., Major, M. C., Orlando B. Mayer, Col., M. C., F.A.C.P., and Robert C. Nelson, Captain, Sn. C., A.U.S.

Recently an impressively high percentage of apparent cures in cases of subacute bacterial endocarditis has been reported by treatment with penicillin. In most of these cases the infecting organism has been a *Streptococcus viridans*. Some strains of this organism, unfortunately have been resistant to penicillin. We wish to report such a case which, although apparently incurable with rather large doses of penicillin, did respond favorably when massive doses of penicillin were employed in the presence of a renal blockage with diodrast and sodium p-aminohippurate. It should be noted that in this case, during the 17 month period in which various doses of penicillin were given, the organism seemed to become increasingly resistant to penicillin.

The method used for determining penicillin sensitivity was as follows: Dilutions of penicillin were set up in (a) thioglycolate broth (Difco) and (b) tryptose-phosphate broth (Difco). A standard inoculum of the streptococcus involved was added to each dilution. End points were determined by subculturing on tryptose agar after 72 hours. It was found that the 72 hour end point was in agreement with subsequent end points determined as long as 10 days after inoculation. The end point used was the smallest amount of penicillin producing negative subcultures after 72 hours incubation of the dilution-culture. The amount required to inhibit growth of the organism was found to be considerably below the lethal requirement. Medium used for blood cultures was Difco tryptose-phosphate broth. Subcultures were made with poured plates of tryptose agar.

The following is a summary of the methods used in determining the concentration of penicillin in blood serum:

1. A solution of penicillin was standardized by the inhibition zone method using the Oxford strain *Staphylococcus aureus*. This solution was used as control for all penicillin serum-level determinations.

2. The determinations were done by two methods: (a) Using Type "O" erythrocytes and a hemolytic streptococcus (Oxford) plus test serum dilutions. The end point for this method was the greatest serum dilution showing no hemolysis of the erythrocytes. (b) Using the Oxford strain *Staphylococcus aureus* in dilutions of the test serum. The highest dilution of serum showing no turbidity was taken as the end point. In both methods as controls known amounts of penicillin were substituted for the serum dilutions and the amount of penicillin in the serum was computed by comparison of the end point dilution

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factors of the control and the test series. In all cases when determinations were made on a serum specimen the results were in agreement.

Case Report

A First Lieutenant bomber pilot, aged 28, first came under our observation on September 2, 1944. The family history was interesting in that his father had died of subacute bacterial endocarditis about five years previously. When the patient was four years of age his family had been informed that he had a systolic heart murmur which was probably not considered clinically significant. There was no history of either rheumatic fever or definite congenital heart disease. The Officer stated that he believes a heart murmur was detected when he entered the service December 7, 1941, but this murmur was again not considered clinically significant. Subsequent examinations during the training period evidently revealed no disqualifying defects and he was commissioned as a pilot June 23, 1942.

On about April 1, 1943, while on duty in North Africa, he first noted the insidious onset of chilliness and fever. During the following month no cause for this fever could be found, but on clinical grounds he was given antimalarial therapy without effect.

On about May 11, 1943 the fever became more marked, accompanied by malaise, generalized aching, and migratory joint pains without any objective evidence of joint disease. The physical examination at that time was normal except for a loud, widely transmitted, harsh systolic murmur at the apex of the heart. He was evacuated to the United States, arriving on August 30, 1943. While in transit he had his first cerebral episode manifested by unconsciousness and a temporary monoplegia of the left arm. On September 22, 1943 a blood culture first became positive for a slow-growing *Streptococcus viridans*. Following a short course of sulfadiazine therapy, he was transferred to another Army hospital where penicillin was available. A note appeared in the clinical record that "fortunately this organism is very sensitive to penicillin", but the evidence for this statement was not further elaborated. During the course of this hospitalization, from November 11, 1943 to August 28, 1944, multiple courses of penicillin therapy were given the patient. His temperature curve usually ranged from 99° to 101–102° F. daily when not under therapy. The various schedules included (a) 120,000 U penicillin IM per day; (b) 200,000 U penicillin IM per day; (c) 200,000 U penicillin IV per day; (d) 100,000 U penicillin plus 100 to 200 mg. sodium heparin IV per day. These schedules all resulted in temporary benefit, but gradually the temperature curve would return to its usual daily swing to 101–102° F. and the blood cultures would remain positive even though under therapy. From November 11, 1943 to August 28, 1944 (when treatment was temporarily stopped) the officer had received a total of 32,000,000 U penicillin. His general clinical condition remained surprisingly good and his spirits optimistic.

At the time of admission to our hospital on September 2, 1944 he presented a picture of a pallid, fairly well nourished, chronically ill individual. The heart was considered slightly enlarged clinically and perhaps by roentgen-ray, and there was a loud, harsh, apical, systolic murmur associated with an easily palpable thrill. Initially we noted rather definite evidence of congestive failure with a gallop rhythm, basilar pulmonary râles, an increase in the subcutaneous fluid, and a persistent hacking cough. These signs disappeared with usual type of therapy and during our first course of penicillin therapy. The blood counts were consistently within normal limits except that the red blood cell count varied between 4.0 and 4.5 million per cu. mm. The urine often showed few to many red blood cells and small amounts of albumin, although it was more usually normal. Electrocardiograms and blood urea nitrogen determinations were always normal. There were many episodes of showers

of petechiae and multiple embolic phenomena of other types including a second temporary hemiplegia of the left arm and leg and a temporary motor aphasia.

From September 2, 1944 to March 12, 1945 the following types of therapy were given with no or but partial and temporary benefit: (a) 240,000 U penicillin IM per day for 30 days; (b) 720,000 U penicillin IM per day for 14 days and 600,000 to 420,000 U penicillin IM for an additional 14 days; (c) 320,000 U penicillin IM per day plus sulfadiazine for 14 days; (d) sulfadiazine alone with blood levels of 10 to 12 mg. per 100 c.c. for 30 days; (e) neoarsphenamine IV according to the schedule of E. E. Osgood [1] for six days. This was discontinued because of an arsphenamine dermatitis. Blood cultures remained positive or were only temporarily reversed, and the temperature curve was only temporarily depressed with all of these regimens.

It was considered on clinical grounds that the organism was probably partially sensitive to penicillin. In November 1944 our first sensitivity test was carried out,

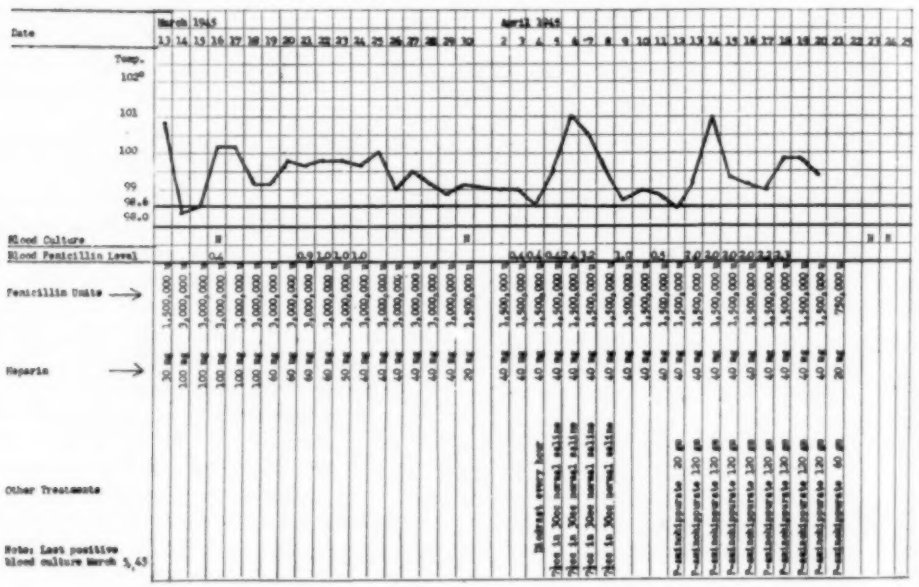

FIG. 1.

revealing the organism to be killed in vitro in a concentration of 0.5 Oxford unit of penicillin per c.c. In March 1945 the sensitivity level had risen to 1.4 Oxford units per c.c. It was then determined to attempt to exceed in the blood by two or three times this theoretical penicillin level, and maintain the level long enough to allow healing of the vegetations. The blood culture obtained March 5, 1945 was reported positive at about this time.

Starting on March 13, 1945 (see table) 3,000,000 U penicillin in 2,000 c.c. of 5 per cent glucose solution per day was given continuously by intravenous drip for 17 days. A small amount of sodium heparin was added to the solution to delay phlebothrombosis and plugging of the needle, but not enough to affect the systemic clotting time appreciably. An average dose of 40 mg. per day was arrived at in this case. The penicillin blood levels and the results of the blood cultures are seen in the table. With the intravenous dosage of 3,000,000 U penicillin, the urine was found to contain in 24 hours approximately 2,000,000 U. There was a remarkable clinical improvement concomitantly, although the temperature curve was not completely normal

and there were still occasional episodes of petechiae. During this 17 day period 51,000,000 U penicillin were given. Since the blood level did not exceed 1.0 unit per c.c., it was believed advisable to employ diodrast as a means of producing a renal blockade.[2]

Accordingly, diodrast was given as follows (see table): Penicillin 1,500,000 U per day IV with 40 mg. of sodium heparin by continuous intravenous drip was given for three days to establish a control blood level, namely 0.4 Oxford unit per c.c. At noon on April 5, 1945 diodrast was added intravenously in the amount of 7.5 c.c. of a 35 per cent solution hourly for a period of 70 hours. The penicillin blood level increased rapidly to 2.4 and 3.2 units per c.c at the end of 24 and 70 hours, respectively. By 24 hours after the diodrast was terminated, the penicillin level in the blood was still 1.0 unit per c.c. The diodrast itself was believed responsible for the temperature elevation to 101° F. on April 6, 1945. No other ill effects were noted.

Through the courtesy of Sharp and Dohme, a supply of sodium p-aminohippurate was obtained, the use of which to produce a renal blockade had been suggested by Beyer and coworkers.[3, 4] Again a three-day control period was run with a continuous intravenous drip of 1,500,000 U penicillin and 40 mg. of heparin, obtaining a constant blood level of 0.5 Oxford unit of penicillin per c.c. A priming dose of 20 grams of sodium p-aminohippurate was given intravenously on the third day and thereafter 120 grams per day were added to the intravenous solution. The blood penicillin concentration rose to 2.0 U per c.c., four times the control level. Again a mild febrile response occurred with the use of this drug, but it soon subsided. The schedule was terminated on April 21 when our supply was exhausted on the ninth day of its use. After this date no further therapy of any kind was given. The temperature curve following April 20 remained quite normal, confirming our clinical impression that the above-mentioned febrile reactions were presumably due to diodrast or p-aminohippurate.

The blood culture obtained on March 5, 1945 was the last one to become positive for the streptococcus. Blood cultures taken on April 23 and 24 and May 4 and 21 were reported negative after 23, 22, 15 and 14 days respectively. From March 13 to April 21 a total of 80,250,000 U penicillin were given. Prior to September 2, 1944, 32,000,000 U had been administered and 39,880,000 U were given by us prior to March 1945 without more than temporary clinical improvement. This made a grand total of 152,130,000 U penicillin throughout.

Comment

It is too early to evaluate this case in terms of a cure. A recurrence with the same or another organism may occur in the future. However, it is noted that after April 21, 1945 the patient was continuously afebrile for the first time in two years, with negative blood cultures since March 5, 1945. He is now ambulatory and shows no signs of activity.

Our plan for penicillin therapy in future cases will be first to determine the specific organism's penicillin sensitivity and then by appropriate means, with or without renal blockage, to obtain penicillin blood levels two or three times those necessary to kill the organism in vitro. It would appear in retrospect that had the plan been attempted earlier, the large amounts of penicillin finally employed would not have been necessary. We believe that these conclusions are valid and offer a rational and concrete approach to penicillin therapy in subacute bacterial endocarditis.

SUMMARY

The plan for penicillin therapy in cases of subacute bacterial endocarditis is presented. Over a period of approximately 17 months a total dosage of 152,130,000 units of penicillin was administered, but apparent cure was not effected until blood levels exceeded by several times the in vitro penicillin sensitivity of the organism. A method is suggested to obtain high serum penicillin blood levels.

ADDENDUM

Subsequent blood cultures as of October 4, 1945 were negative. Following increased physical activity the heart became noticeably enlarged, but no recurrence of the endocarditis has been observed.

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A CASE OF LYMPHOGRANULOMA VENEREUM ASSOCIATED WITH ATYPICAL PNEUMONIA *

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LYMPHOGRANULOMA venereum is no longer thought of as a local infection of the genitalia, regional lymphatics, rectum, and sigmoid, but as Harrop [1] has described, a systemic infection capable of producing headache, septic fever, chills, sweats, and articular rheumatism, and capable of involving lymphatics other than those of the inguinal and pelvic regions. The patient reacts to this infection with an elevation of serum globulin, thought by Schamberg [2] to be an evidence of humoral antibody response. The skin becomes hypersensitive to the intradermal injection of suspensions of the inactivated virus, the Frei test.

Rake and his coworkers [3] have improved the Frei antigen test for this disease, and in addition have introduced a complement fixation test, as well as a specific antitoxic reaction.[4] The same workers [5] have described similarities and discussed relationships among the viruses of lymphogranuloma venereum, psittacosis, atypical pneumonia and meningopneumonitis.

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When mice are inoculated intracerebrally with the virus of lymphogranuloma venereum, meningitis results. That this experimental finding applies to man was recently shown by Sabin and Aring,[6] who conclusively demonstrated the occurrence of meningoencephalitis in man due to the virus of lymphogranuloma venereum.

When the virus of lymphogranuloma venereum is inoculated intranasally into mice, pneumonia results. The pathological picture in the lungs is quite like that seen in the so-called "virus" or atypical pneumonia of man.[7] The same group of viruses noted above is believed to cause atypical or "virus" pneumonia in human beings.[8]

From the foregoing one might expect to encounter pneumonitis in some cases of lymphogranuloma venereum. We know of no reference in the literature describing this association in man.

The following case report is presented only because of the concurrent presence of both lymphogranuloma venereum and atypical pneumonia.

Case Report

A 29 year old colored sergeant was transferred to Percy Jones General Hospital on February 17, 1943. He had been quite well in the past except for an episode of gonococcic urethritis one year before, which had responded promptly to treatment. Family history revealed that his mother had died of tuberculosis some years before. During the month of December 1942 he began to note a hacking cough which had never disappeared. He had no other complaints until he returned from a furlough on January 3, 1943. On this date he began to feel badly, and two days later entered the station hospital at his camp, because of malaise, cough and chills. On admission, his temperature was 104° F., pulse 88, and respirations 24. Physical examination at this time was negative except for impairment of percussion at the right base and roentgenographic evidence of an elevated right diaphragm, but no pulmonary disease. For the next few days, his condition remained essentially the same, his temperature staying at about 102° F. On January 8 he developed severe pleuritic pain on the right and by the next day a pleuritic rub was in evidence as well as bronchial breathing and râles at the right base. Roentgenogram now showed in addition to the elevated right diaphragm, what was interpreted to be pneumonitis (figure 1). Sulfathiazole was administered between January 9 and January 15, with no apparent effect. His temperature gradually came down to normal by January 14 and the white blood count, which had been 18,700 on January 11 soon dropped to 13,400.

During the next few weeks he felt fairly well and was up and about. His only complaint during this time was aching pain in the right axillary region. The roentgenogram now showed gradual clearing, with an area of plate-like atelectasis (figure 2.) On about February 5 he became aware of a somewhat tender swollen lymph gland in the right axilla, which subsided spontaneously within a week. This was followed by gradual enlargement of the lymph glands in the left inguinal region, first noted on February 12. There was slight elevation of temperature at this time. As it became apparent that he was not recovering promptly, he was transferred to the Percy Jones General Hospital on February 17. On arrival, his temperature was 101° F., and he appeared somewhat prostrated. Moderate bilateral axillary adenopathy was present. An enlarged, moderately tender, almond sized node was felt in the left inguinal region. The remainder of the physical examination was negative except for some limitation of motion of the right thorax and signs suggestive of elevation of the right diaphragm.

The initial laboratory studies, including urinalysis, Kahn reaction, blood culture, agglutination for *B. tularense* as well as culture of lymph from the left inguinal node, obtained by aspiration, were all negative. Red blood cell count and hemoglobin reading were normal but the white cell count was elevated to 12,500 with a normal differential. Blood sedimentation rate was 84 mm. per hour. His temperature, which averaged 102° F. for the first several days after transfer, gradually subsided to normal by March 6. On March 8 the left inguinal gland which had enlarged to the size of a pecan was removed for biopsy. Following this procedure, low grade fever was present for a few days. The wound drained for a time and then gradually healed.

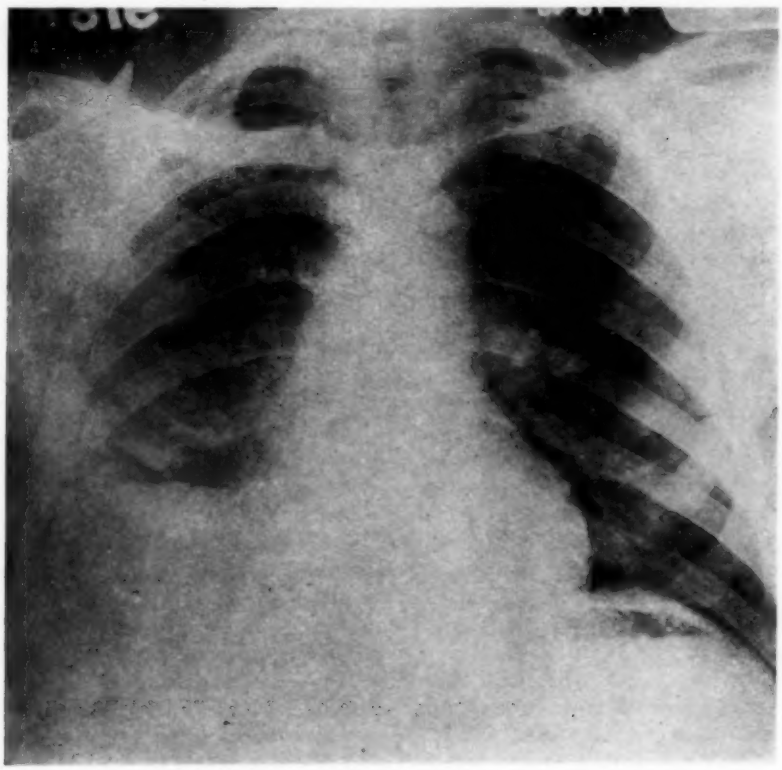

FIG. 1.

The remainder of his hospital course was uneventful except for an episode of cellulitis adjacent to the wound which developed on March 27. This was treated with sulfadiazine and the cellulitis and accompanying fever disappeared within a few days. The sulfadiazine was continued for a period of four weeks and blood levels of 5 to 7 milligrams per 100 c.c. were obtained. All traces of pleural and pulmonary involvement gradually disappeared, only a few adhesions to the diaphragm remaining. There was no remaining adenopathy of note. Because of administrative reasons he was kept in the hospital until August. After a final proctoscopic examination which showed the rectum and sigmoid to be entirely normal he was discharged to duty.

Laboratory. Serum albumin was 4.5 and globulin 4.7 grams per 100 c.c. The Frei test was positive repeatedly. Blood sedimentation rate gradually dropped to

normal. Section of the gland which was removed revealed it to be honeycombed with soft yellowish accumulations of pus. Cultures, smears and guinea pig inoculations, as well as tissue stains for organisms, were performed with negative results. The microscopic sections were suggestive of lymphogranuloma venereum. Serum was sent to the laboratory of Dr. Geoffrey Rake, of the Squibb Institute for Medical Research, for complement fixation tests with the virus of lymphogranuloma venereum and related viruses. The patient's serum fixed complement in high dilution with the specific virus, and in much lower dilutions with the other viruses of this group. Likewise, the serum exhibited the recently described specific antitoxic reaction. These data convinced us that we were dealing with an acute case of lymphogranuloma venereum and that possibly the one agent was the cause of both pathologic processes.

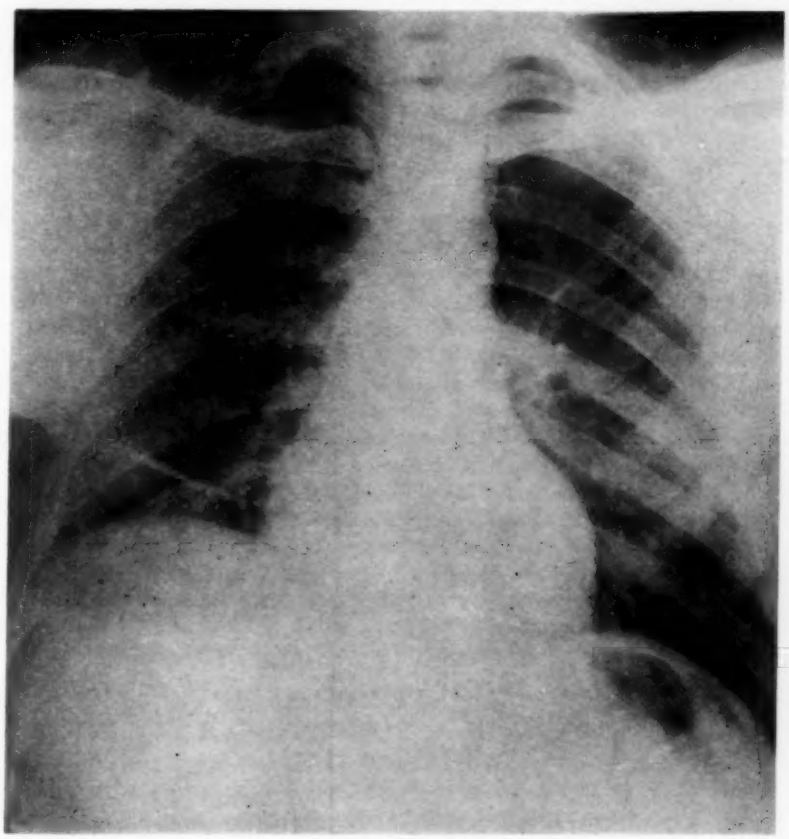

FIG. 2.

SUMMARY

A case exhibiting pneumonitis and lymphadenopathy accompanied by laboratory evidence of lymphogranuloma venereum is presented. It is suggested that the combination of pathologic processes may have been due to the virus of lymphogranuloma venereum.

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A CASE OF CORONARY THROMBOSIS WITH MYOCARDIAL INFARCTION IN A NINETEEN YEAR OLD WHITE MALE *

By GUY A. RICHARDS, M.D., *Washington, D. C.*

CORONARY artery disease exists in young people. Glendy, Levine, and White [1] have reported a series, the youngest patient being 20 years old. Recently French and Dock [2] reported a group of men 20 to 36 years of age all of whom had proved coronary thrombosis with myocardial infarction.

A case of coronary thrombosis in a 19 year old youth is reported.

CASE REPORT

A patient aged 19 years was admitted to Emergency Hospital January 10, 1945, complaining of severe constant pain radiating across the abdomen just above the umbilicus.

* Received for publication April 6, 1945.

Eighteen hours prior to admission the patient awoke with abdominal pain described as constant, severe, with no tendency to wax or wane, and not altered by change in position. Prior to retiring the night before, he had felt well following attendance at a party where he ate heartily of cheese, hamburgers, and fruit cake. There was no nausea or vomiting associated with the pain. He had a normal bowel movement in mid-morning. He was not aware of having fever, chills or shortness of breath, but he noted that he had not voided all day.

Late in the afternoon he was visited by a physician who made a diagnosis of acute gastroenteritis and prescribed an antacid which he promptly vomited. An enema was given with good results.

In the past he had had measles, mumps, chickenpox, an appendectomy at the age of five, and scarlet fever at the age of 15. There was no history suggestive of rheumatic fever. Review of systems was completely negative except for strabismus convergens present from birth, for which he was rejected from the armed services.

The family history was entirely non-contributory.

On physical examination he appeared acutely ill, his temperature was 99.4° F., pulse rate 96 per minute, respiratory rate 30 per minute, blood pressure 128 mm. Hg systolic and 84 mm. diastolic. His skin was flushed, warm and moist. There was strabismus convergens of the eyes. The pupils were equal and reacted to light and accommodation. Teeth, tonsils, nose, and throat were normal and there was no rigidity or lymphadenopathy of the neck. The chest was clear to percussion and auscultation. The heart sounds were distant, but quite distinct. Rate was 96 per minute, and rhythm was regular. No murmurs were heard. Tenderness and rigidity of the abdomen were marked; this was more severe in the epigastrium, and there was no localized point of tenderness. There was no referred or rebound tenderness, and no palpable masses. Bilateral costovertebral tenderness was present and percussion over the kidneys produced waves of pain across the abdomen. Peristalsis was active. The extremities were normal and the neurological examination negative.

The hemogram on admission was as follows: Hemoglobin 14 grams per cent or 85 per cent, red blood cells 4,650,000 per cu. mm., white blood cells 26,000 per cu. mm. with a differential count of 95 per cent polymorphonuclear neutrophiles, 4 per cent lymphocytes and 1 per cent monocytes. The urinalysis revealed: color amber, slightly cloudy, acid in reaction, specific gravity 1.010, heavy trace of albumin, and negative sugar reaction. Microscopic examination showed numerous epithelial cells, red and white blood cells.

The preliminary diagnosis at this time was: (1) Acute glomerulonephritis; (2) peritonitis; etiology: (a) perforated ulcer; (b) pneumococcal peritonitis.

Treatment was symptomatic and palliative.

The next morning the patient appeared to be worse, the temperature was 101.2° F., pulse rate 120 per minute, respirations were 30 per minute and very labored. The blood pressure was 124 mm. Hg systolic and 80 mm. Hg diastolic. He described the pain as just the same although morphine sulfate gr. ¼ had given him relief. The physical signs were unchanged except it was believed that tubular breathing was heard over the right upper lobe of the lung posteriorly.

Roentgenograms of the chest and abdomen revealed no abnormality. The hemoglobin and red cell count were unchanged, but the white cells had increased to 35,000 with a differential of 92 per cent polymorphonuclear forms, 7 per cent lymphocytes and 1 per cent monocytes. The blood amylase was 142 units (Somogyi, normal 80 to 150). The non-protein nitrogen blood level was 64 mg. per cent. The urinalysis was essentially the same, with a large amount of albumin present and red cells and bacteria in large numbers.

Late in the afternoon the patient complained of pain, burning in character, in both heels, but both feet were warm and of normal color, and the pulsation of the dorsalis pedis arteries was normal. No evidence of thrombosis, either arterial or

venous, could be found. The temperature, pulse, and respirations remained the same.

During the night of January 11, the patient slept at intervals, requiring morphine sulfate gr. ¼ twice to relieve the pain. On January 12, at 7:30 a.m., he stated that the pain was less severe and that he felt better, although it was noted that he was very pale and slightly cyanotic. Oxygen was started. The temperature was now 101.4° F., pulse 108 per minute, and respirations were 28 per minute. By 8 a.m. he was markedly cyanotic and respirations ceased suddenly at 8:15 a.m.

At the postmortem examination the pathological findings were confined to the heart, kidneys and lungs.*

The pericardial sac contained 3 c.c. of clear, colorless fluid. The heart was enlarged slightly; the surface was smooth and glistening. All the valves were grossly normal. The tip of the left ventricle contained a small intramural thrombus. The descending branch of the left coronary artery contained a well organized thrombus one and one half centimeters in length, within one and one-half centimeters of the origin of the vessel. The heart muscle was mottled, and several yellowish to white areas were noted in the left ventricle. Examination of the interventricular septum revealed a typical myocardial infarction.

Both the lungs hung free in the pleural cavity. The lower right lobe was atelectatic posteriorly, and the remainder was crepitant. The left lower lobe was also atelectatic, and on section moderate edema was present.

Both kidneys were hemorrhagic in appearance, slightly swollen, and contained multiple small early infarcts. On section there was evident extensive acute necrosis due to multiple emboli. The left renal artery was filled with an organized embolus.

Microscopically the heart muscle fibers were of average size, they stained unevenly, and marked fragmentation was noted. There was an advanced degree of acute necrosis of the muscle, and all the vessels were filled with fresh thrombi, and some partially organized thrombi. The muscle tissue was heavily infiltrated with polymorphonuclear cells and large histiocytes containing cellular debris. Hemorrhage was present in some portions.

The thrombosed coronary artery was severely diseased. There was advanced cystic degeneration of the media with a large plaque of atheromatous deposit within the media. The intima was denuded, and part of the adherent thrombus was present. The periarterial tissue was edematous and infiltrated by red blood cells, lymphocytes and plasma cells.

Section through the mural thrombus showed it to be a well organized clot with dense bands of fibrin, and infiltrated with large mononuclear cells, lymphocytes and polymorphonuclear cells.

The lungs were intensely congested. The alveoli were partially atelectatic and contained many "heart failure cells."

The kidney parenchyma was widely destroyed as a result of multiple hemorrhagic infarcts. Many vessels were filled with fresh thrombi, and a few contained portions of organized emboli. One fair sized artery was completely occluded by an organized embolus which had become incorporated with the vessel wall. The infarcted tissue was hemorrhagic and heavily infiltrated by polymorphonuclear cells, and the intervening tissue was completely necrotic. It was also noted that those glomeruli which had not undergone acute infarction or secondary necrosis were badly fibrosed, and there was an unusual degree of sclerosis of the afferent arterioles. Some of the other arterioles were completely occluded and others narrowed. This was a most severe degree of arteriolarsclerosis.

The final diagnosis was: (1) Acute coronary thrombosis with myocardial infarction; (2) severe coronary atheromatosis with medial cystic degeneration; (3) multiple renal infarcts; (4) marked arteriolarsclerosis.

* Postmortem examination by Dr. Charles Eronstein.

SUMMARY

A case of coronary thrombosis with coronary artery sclerosis, renal infarction and arteriolarsclerosis, with postmortem findings, in a 19 year old white male is reported.

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PAROXYSMAL VENTRICULAR TACHYCARDIA ASSOCIATED WITH SHORT P–R INTERVALS AND PROLONGED QRS COMPLEXES *

By MORRIS E. MISSAL, Lt. Colonel, F.A.C.P., DOUGLAS J. WOOD, Captain, and SIDNEY D. LEO, Captain, M.C., A.U.S.

ALTHOUGH earlier writers,[1, 2, 3] had discussed the syndrome of short P–R intervals and widened QRS complexes, proper clinical evaluation of the problem was not accomplished until the paper of Wolff, Parkinson, and White,[4] by whose names the syndrome is frequently known. These earlier papers referred to the relative frequency of associated paroxysmal auricular tachycardia or auricular fibrillation.

Recently interest has been renewed in the syndrome because of its occurrence with paroxysmal ventricular tachycardia. The first report apparently was that of Arana and Cossio in 1938,[5] whose patient had episodes of auricular fibrillation and paroxysmal ventricular tachycardia associated with this syndrome. In 1940 Hunter, Papp, and Parkinson [6] described two patients in whom the syndrome occurred with paroxysmal ventricular tachycardia; one of the patients subsequently developed a supraventricular tachycardia, the other auricular fibrillation. Three additional cases of Wolff-Parkinson-White syndrome and paroxysmal ventricular tachycardia were described by Levine and Beeson [7] in 1941. A more recent report by Palatucci and Knighton [8] describes a similar case. Many of the above writers comment on the difficulty of excluding recent myocardial infarction in some of these patients.

The patient herein described represents another example of paroxysmal ventricular tachycardia associated with short R–R intervals and wide QRS complexes. Certain features of this case would seem to justify the addition of another report to the literature.

CASE REPORT

A 20 year old flying officer was admitted to the hospital on February 29, 1944 complaining of a "grabbing" pain in the epigastrium and sternum which was aggravated by effort and diminished by rest.

* Received for publication April 14, 1945.
From the Medical Service, AAF Regional Station Hospital, Army Air Base, Richmond, Virginia.

The officer presumably had been in his usual good health until approximately 66 hours prior to admission. During his training, he had successfully passed three of the routine ("type 64") physical examinations given flying personnel. The last of these examinations had taken place five days before admission. Electrocardiographic examinations had not been included.

Three days prior to admission, the pilot had reported for a routine "flight" in the low-pressure chamber. Along with others, including a trained observer, he "ascended" to a simulated height of 18,000 feet without the use of accessory oxygen. This procedure is a routine one to acquaint each flyer with his own reaction to anoxia.* Constant supervision is maintained. Only the usual mild symptoms were experienced by the patient, i.e., "heavy breathing" and a lack of alertness. No vertigo or visual difficulties were experienced. Individually fitted masks permitting inhalation of 100 per cent oxygen were then applied and used at this altitude for 15 minutes. The group, continuing to breathe pure oxygen, was then taken to a simulated altitude of 38,000 feet.† This elevation was maintained for 16 minutes, during which the subject noted only slight abdominal discomfort. During the "descent," however, he experienced moderate difficulty in adequately ventilating his middle ears, a not unusual symptom. Upon reaching "ground level" the patient felt well except for a slight sensation of blockage of the right ear. There was otoscopic evidence of a mild bilateral aero-otitis media (mild injection of both tympanic membranes, without edema or retraction).

At the termination of the above proceedings, the patient left the base to attend a celebration which lasted throughout the night. Three friends and he consumed two quarts of whiskey and smoked many cigarettes. Returning to quarters at seven the following morning, he felt ill and vomited several times. After four hours of sleep, he awakened conscious of tachycardia, anorexia, and malaise. He remained in or around his quarters all that day and the following day, during which he continued to suffer from malaise and anorexia. He was aware of a rapid pulse rate most of the time. There is no record of the pulse rate or heart rhythm during this period.

On the following day, approximately 60 hours after the completion of the "chamber flight," the patient noted the gradual onset of a "grabbing" sensation in the epigastrium. This was increased by walking and alleviated by resting. Although he was tired, there was no complaint of dyspnea. Upon reporting to his flight surgeon approximately 66 hours after the "flight," he was immediately admitted to the hospital.

On admission the patient was acutely ill and showed moderate perspiration and cyanosis. His respirations were normal in character and the rate was 18 per minute. The pulse was irregular and difficult to count. The blood pressure was determined as 110 mm. Hg systolic and 70 mm. diastolic. The temperature was 98.4° F. Prominent, irregular jugular pulsations were noted, but there were no cardiac pulsations or thrills. The heart was of normal size on clinical and radiologic examination. No murmurs or friction rubs were heard. A totally irregular rhythm was present, and sounds were of poor quality. The cardiac rate, difficult to ascertain, was counted as approximately 150 per minute. A gallop was not detected. There was evidence of marked right-sided heart failure as shown by a tender, non-pulsating, enlarged liver, increased

* An altitude of 18,000 feet, actual or simulated, represents an atmospheric pressure of 379.4 mm. Hg, or one half the standard pressure at sea level. The partial pressure of oxygen in the atmosphere at this altitude is 79.4 mm. Hg. At 18,000 feet the alveolar oxygen tension of an individual not breathing supplementary oxygen would be approximately 42 mm. Hg; his oxyhemoglobin saturation would be approximately 71 per cent.

† The atmospheric pressure at an altitude of 38,000 feet, actual or simulated, is 154.9 mm. Hg. The partial pressure of oxygen at this altitude is 32.4 mm. Hg. An individual breathing 100 per cent oxygen under these conditions would have an alveolar oxygen tension of 72 mm. Hg; his oxyhemoglobin saturation would be approximately 90 per cent.

venous pressure, and evidence of a small left pleural effusion. No peripheral edema was present.

The following facts pertaining to the past history were elicited later from the patient and his mother: questionable scarlet fever in childhood, frequent sore throats until the age of 12, and several unexplained nose bleeds in 1941. At no time had there been joint pains or chorea. At the age of 15, fainting spells had occurred when the

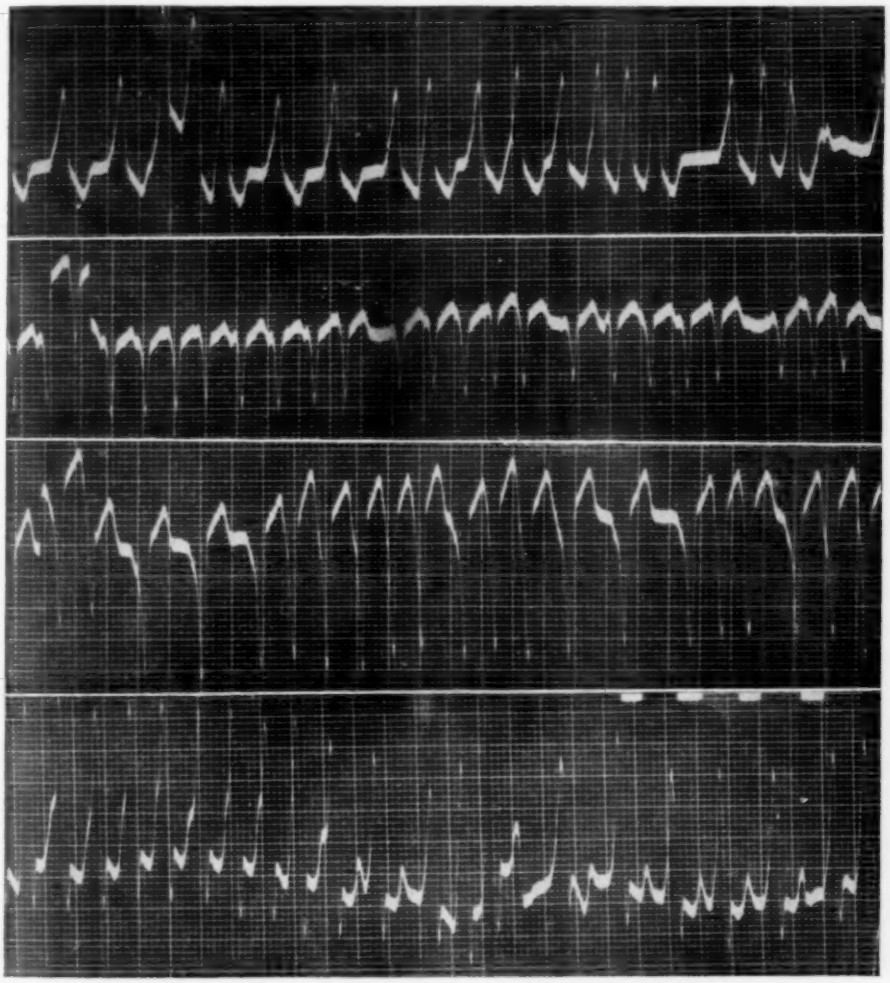

FIG. 1. Initial electrocardiograms, taken within one hour of admission. Ventricular tachycardia, rate varying between 230 and 250.

patient played basketball or football, and on one occasion he had been directed by his doctor to decrease his activities for a week because he had "strained" his heart. No further details of these episodes were available. Both a maternal and a paternal uncle had died of heart disease of unknown types.

Electrocardiographic Studies and Course. Figure 1 represents the initial electrocardiogram, taken within an hour of admission. This shows irregular ventricular

tachycardia with a rate varying from 230 to 250. Frequent pauses are noted. In Lead II the slight deflection preceding the main downward deflection is interpreted as part of the QRS complex rather than the P-wave it resembles at first glance. Carotid sinus pressure failed to affect either the rate or rhythm. An electrocardiogram taken one hour later showed no significant changes. (Times identifying the various electrocardiograms are in terms of the 24 hour clock.)

One hour before electrocardiogram 2A was taken a 0.2 gm. test dose of quinidine sulfate had been administered by mouth. This was followed by 0.4 gm. every two hours.

Electrocardiogram 2B, taken after a total dose of 1.0 gm. of quinidine, shows restoration of sinus rhythm, and was the first one showing short PR intervals and wide QRS complexes (Rate 100; PR .10–.12 sec.; QRS .12 sec.). A ventricular premature contraction is seen in Lead CF4.

Only minor variations are noted among tracings 2B, 2C, and 2D. However, in electrocardiogram 2E, taken approximately 17 hours after admission and 13 hours after initiation of quinidine therapy (total dosage 2.6 gm.), a striking change may be observed, i.e., prolongation of PR to .16 sec., diminution of QRS to .08 sec., and inversion of T_2 and T_3. Five hours later the spontaneous return of the earlier pattern of short PR interval and widened QRS complex was noted (electrocardiogram 2F). At this time quinidine dosage was reduced to 0.2 gm. four times a day and maintained thus until April 25. On March 15 (electrocardiogram 4F) an attempt was made to reduce quinidine to three doses a day but because numerous premature contractions were noted, the dosage was returned to the previous schedule. On May 1 quinidine was discontinued. Electrocardiograms have continued to show the short PR: wide QRS pattern.

Electrocardiograms 3A to 3F demonstrate almost daily variations in the T-waves and a persistence of the short PR: wide QRS pattern, while electrocardiogram 4A (March 9) once more showed the spontaneous occurrence of normal PR intervals and QRS complexes. This electrocardiogram is almost identical with electrocardiogram 2E (March 1). Variations in the configuration of T-waves are noted in electrocardiograms 4B to 4G. Ventricular premature contractions are observed in the last two tracings of this series.

Following subsidence of the ventricular tachycardia, the signs of heart failure diminished markedly within 24 hours. The liver rapidly shrunk in size, and its tenderness disappeared. Within a few days there were no signs of the small pleural effusion.

Shortly before the patient's discharge from the hospital, and at a time when the electrocardiogram showed normal sinus rhythm with the Wolf-Parkinson-White syndrome, carotid sinus pressure produced no changes in the tracing. Subcutaneous and intravenous atropine each effected a decrease in the QRS complex from 0.13 to 0.08 sec.

On the thirty-ninth hospital day, the patient was permitted to sit up in a chair and from that time on his activities were gradually increased. Quinidine was discontinued on the sixty-first day. The patient remained afebrile throughout his entire hospital stay. He was discharged on the eighty-fifth day.

The following laboratory findings are significant. With the exception of a leukocyte count of 10,800 on admission, and one of 9,200 several weeks later, all white counts were from 5,100 to 5,900. The erythrocyte and differential leukocyte counts, as well as hematocrit determinations, were within normal limits. Urinalysis and blood non-protein nitrogen determinations were also normal. Erythrocyte sedimentation rates by the Wintrobe technic were within normal limits (0–3.5 mm. per hr.). The Kahn reaction was negative. All radiologic studies of the chest, including careful fluoroscopy, were negative for evidence of heart disease.

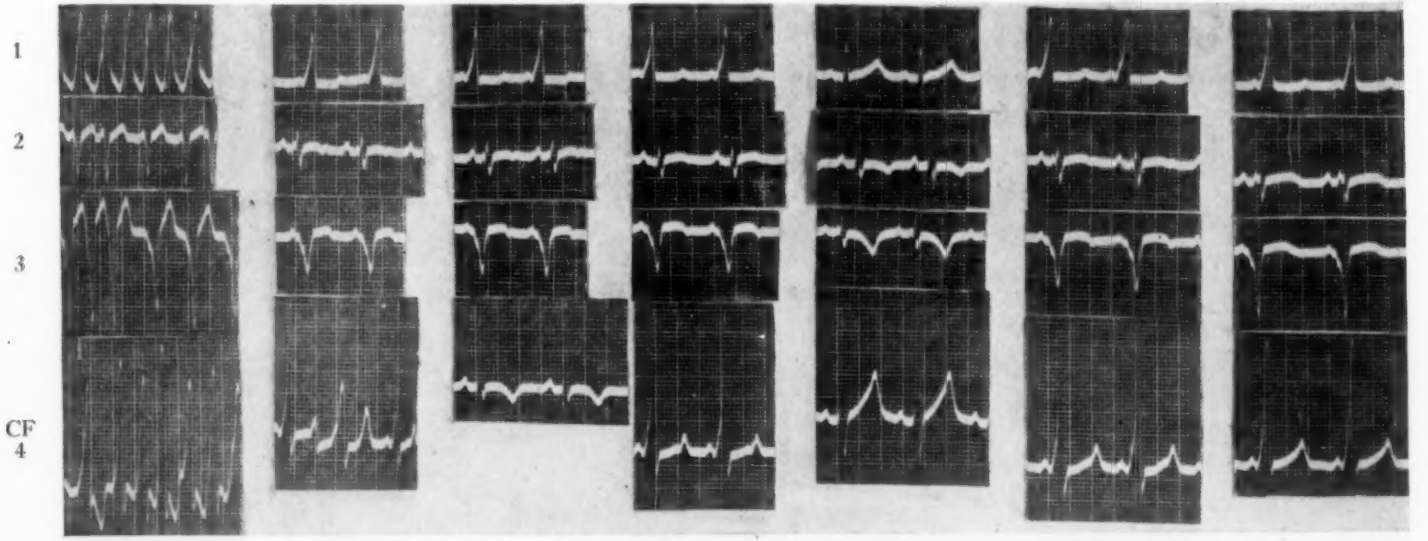

FIG. 2. Tracing A shows no significant change. Note restoration to sinus rhythm, short PR interval and widened QRS complex in B. C and D show only minor differences from B. In tracing E, PR is prolonged to .16 sec. and QRS diminished to .08 sec.; T_2 and T_3 are inverted. Tracings F and G show reversion to short PR and prolonged QRS.

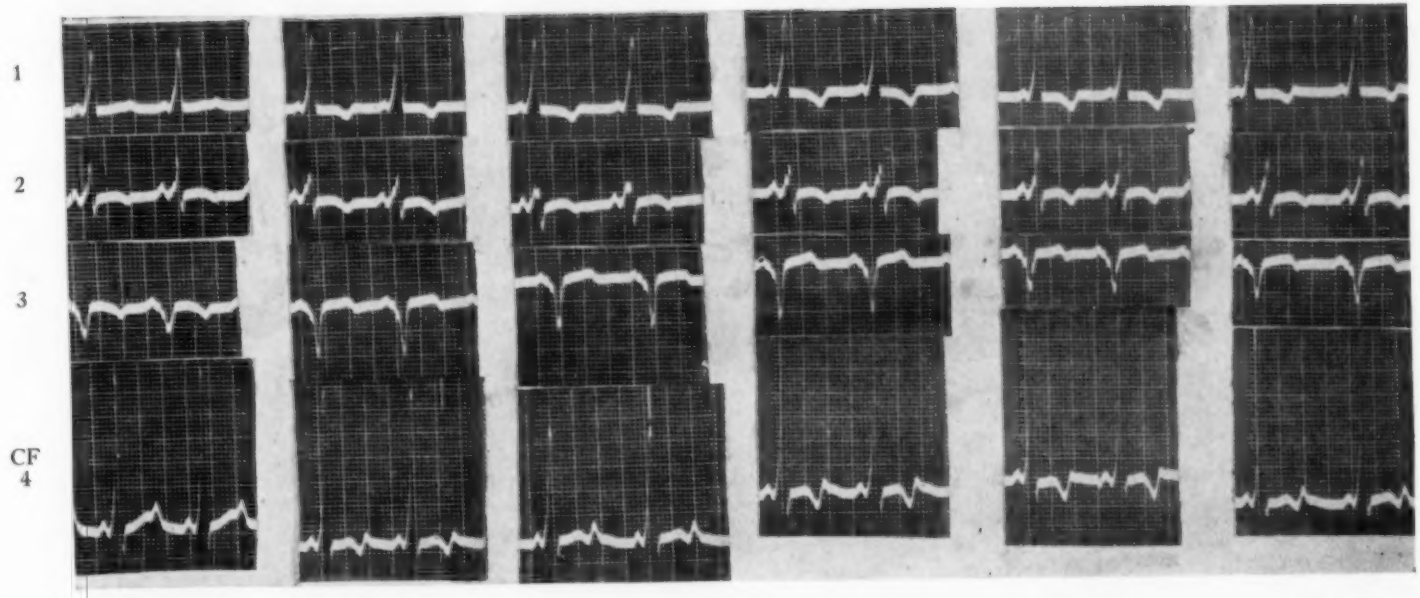

FIG. 3. Persistence of short PR and wide QRS. Striking variation in configuration of T-waves. Note prolonged Q–T interval.

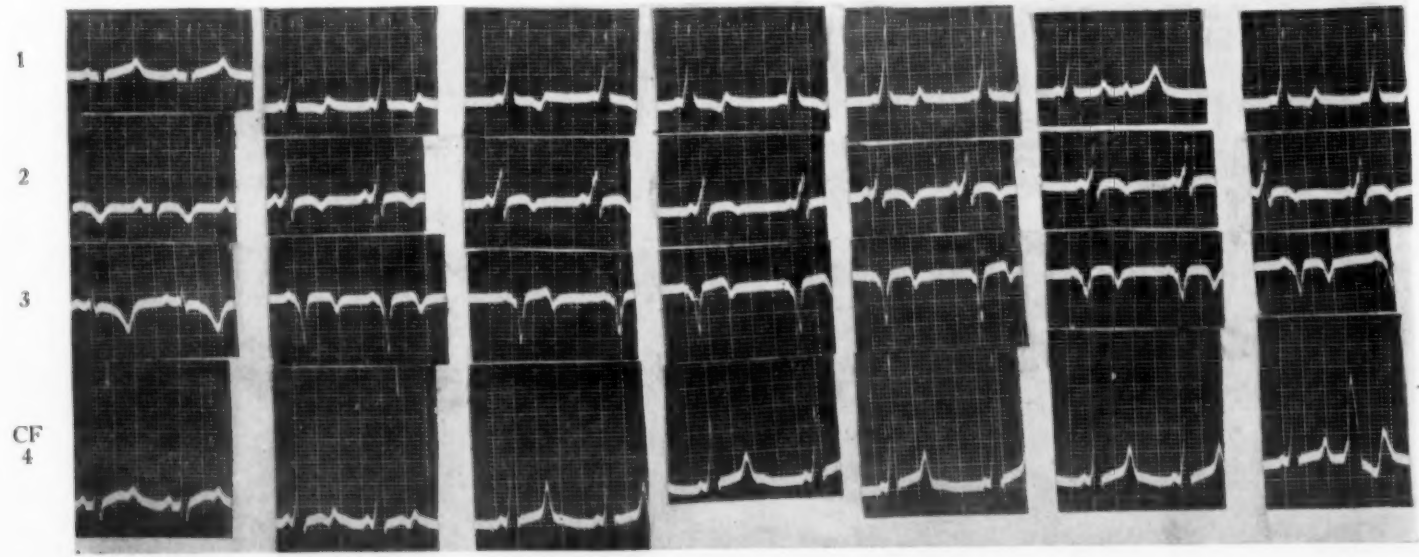

FIG. 4. Spontaneous return to normal PR and QRS. Remainder of tracings show prolonged PR interval, widened QRS complex, and variations in T-waves. Note ventricular premature contractions in tracings F and G.

Discussion

The authors interpret the arrhythmia as irregular paroxysmal ventricular tachycardia. Because there is some similarity in the form of ventricular complexes before and after cessation of the abnormal rhythm, and because the nature of the auricular rhythm is uncertain, the disturbance also could be interpreted as that of auricular fibrillation with aberration of intraventricular conduction.

Paroxysmal ventricular tachycardia is a disturbance of cardiac rhythm almost invariably associated with serious organic heart disease. The patient described here cannot be said, with any degree of certainty, to have had a normal heart prior to this episode. In addition to the occurrence of fainting spells during adolescence and a family history of heart disease, the history of nose bleeds and frequent sore throats is suggestive of stigmata of rheumatic fever.

Outstanding among the possible precipitating factors leading to the paroxysmal arrhythmia would appear to be the consumption of an inadvisable quantity of alcohol in combination with large amounts of tobacco and loss of sleep. The interval of 14 hours transpiring between the completion of the chamber "flight" and the onset of subjective tachycardia makes it unlikely that exposure to reduced atmospheric pressure contributed significantly to the disturbance of cardiac rhythm.

There does not appear to be sufficient evidence for the diagnosis of myocardial infarction, though for a time this was suspected. T-wave changes of the type seen in these tracings often are observed after prolonged episodes of tachycardia and generally are attributed to anoxemia brought about by the rapid cardiac rate. The persistence of T-wave abnormalities beyond the usual period following the reversion from tachycardia to normal sinus rhythm renders this explanation not entirely unsatisfactory. Though myocardial disturbances of some kind probably were present, accurate delineation of the process is difficult.

The original paper of Wolff, Parkinson, and White [4] presented the syndrome of short PR intervals and widened QRS complexes as occurring in young people with otherwise normal hearts who are subject to attacks of paroxysmal arrhythmia. Hunter et al.,[6] in a series of 22 patients (including three with short PR and normal QRS), presented these interesting facts: a short PR: wide QRS pattern occurred in 5 per cent of 140 consecutive cases of bundle branch block and in about 5 per cent of all patients subject to paroxysmal tachycardia. Although most observers consider that the majority of patients with Wolff-Parkinson-White syndrome have no underlying organic cardiac disease, one must bear in mind that heart disease may be present in some.[6] Eighteen of the 90 cases reviewed by these authors and three of their own 19 patients had organic heart disease. Occasionally the causal connection seems to be definite. In one of their patients the characteristic electrocardiogram occurred soon after rheumatic fever. Examples also have been reported following coronary occlusion.

Even in a patient with associated heart disease, prognosis is usually unaffected by the syndrome, though occasionally patients die during the paroxysms of tachycardia. Where the condition is produced by heart disease, apparently a relatively uncommon happening, the prognosis rests upon the nature and degree of the underlying cardiac lesion. These patients may become seriously ill during

the various arrhythmias. Such was the case with the patient who is the subject of this report.

Although it is not the purpose of this paper to enter into any controversy about the mechanism of the Wolff-Parkinson-White syndrome, a few comments may be offered concerning present day concepts of the condition.

Wolferth and Wood,[9] as well as Holzmann and Scherf,[10] suggested that the mechanism was ventricular asynchronism with premature stimulation of one ventricle. These authors assumed that the paroxysmal tachycardia was due to retrograde conduction over the Bundle of Kent or a similar structure causing a reëntry phenomenon in the auricles, with the production of tachycardia. Such connections have been demonstrated anatomically between the auricles and ventricles,[11] and confirmatory evidence obtains from the careful experiments of Butterworth and Poindexter.[12] By using an amplifier to produce an abnormal pathway between the auricles and ventricles, these workers were able to stimulate one ventricle six to eight hundredths of a second sooner than the other. They were able thereby to produce electrocardiograms comparable to those seen clinically. In addition, these workers were able to produce paroxysmal supraventricular tachycardia by reversing the flow of current in the abnormal conduction pathway.

Recognition of the short PR: wide QRS syndrome is not of mere academic interest, since confusion may occur with true bundle branch block or with myocardial infarction. The syndrome is characterized by upright P-waves when the rate is normal, abolition of the isoelectric segment from P to R, and reduction of the PR interval to 0.12 sec. or less. The ventricular complex resembles that of bundle branch block. It is widened beyond 0.10 sec. and often slurred in its ascent and notched near its summit. The RT period may be depressed or elevated, seldom assuming the full diphasic characteristic of ordinary bundle branch block. There also is a tendency for the PT interval (beginning of the P-wave to the end of the QRS complex) to remain constant when normal and abnormal complexes are compared in the same person.

Another interesting feature is that the same patient may show abnormal complexes on one occasion and normal ones on another, the rate being normal on both occasions.[14] Such changes may occur spontaneously or may be induced by exercise or atropine.

Summary

A case of irregular paroxysmal ventricular tachycardia associated with short PR intervals and wide QRS complexes (Wolff-Parkinson-White syndrome) is reported. During the arrhythmia, the patient developed signs of right heart failure. Quinidine produced an excellent therapeutic response. Pertinent literature is discussed.

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A CASE OF SHORT PR INTERVAL AND PROLONGED QRS COMPLEX WITH A PAROXYSM OF VENTRICULAR TACHYCARDIA *

By Max J. Klainer, Captain, M.C., A.U.S., *Stoneham, Massachusetts,* and Harold H. Joffe, Captain, M.C., A.U.S., *Duluth, Minnesota*

In 1930 Wolff, Parkinson and White called attention to a syndrome of "bundle branch block with short PR interval" occurring in healthy young people who were prone to recurrent attacks of paroxysmal auricular tachycardia or auricular fibrillation.[1] In recent years this syndrome has been recognized with increasing frequency and it has been established that this was not a bundle branch block, but the manifestation of an early arrival in the ventricle of the auricular impulse through an accessory pathway, the bundle of Kent.[2, 3] In 1941 Levine and Beeson called attention to the fact that this syndrome could also be associated with paroxysms of ventricular tachycardia.[4] They commented that only two such cases had been previously reported and added three more. A further review of the literature on this subject failed to reveal any additional cases. The purpose of this paper is to add one more case to this small series.

* Received for publication May 5, 1945.

Case Report

A 29 year old white male with one month of military service was admitted to the Regional Hospital, Fort Riley, Kansas, on February 10, 1945, complaining of rapid irregular heart action. Past history revealed that since the age of 19 this patient had been subject to recurrent bouts of this nature. He had had about 18 such attacks which were from one to five days in duration. At the age of 20 he was kept in bed for six weeks following such an attack. In June 1944 an electrocardiogram was taken during a paroxysm of tachycardia and he was treated with quinidine. The onset of each episode was sudden and was accompanied by vertigo, dyspnea and an aching

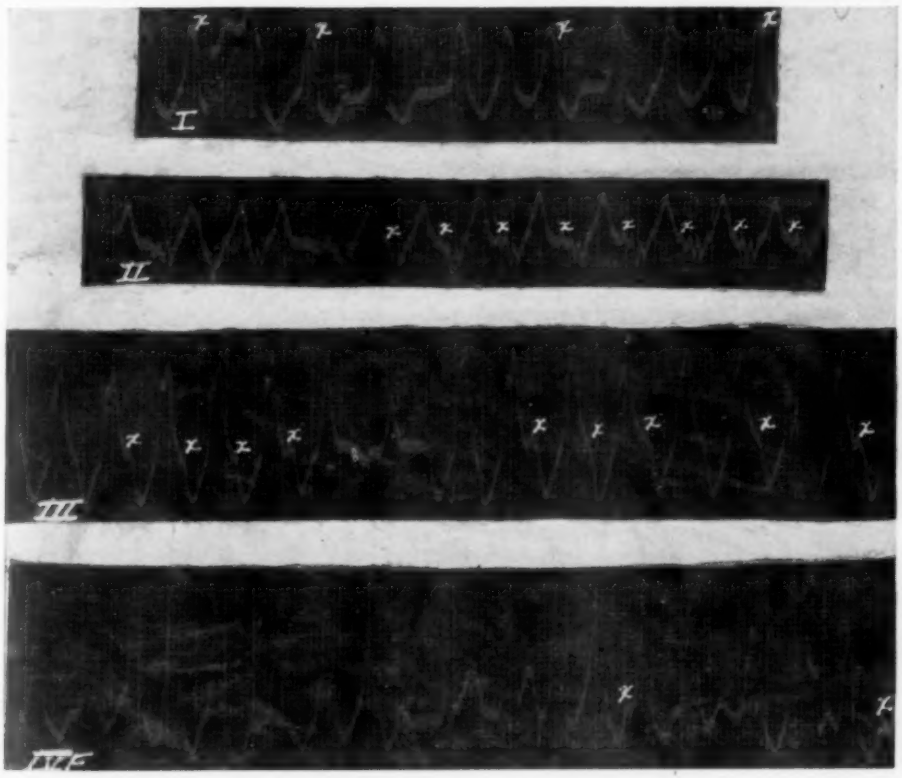

Fig. 1. Electrocardiogram taken on admission during the paroxysm of tachycardia and interpreted as ventricular tachycardia. The QRS complexes are slurred and widened in all leads with a maximum duration of 0.16 second. The ventricular rate is 150 beats per minute. The T-waves are opposite the main deflection in all leads. There are occasional supraventricular beats. The auricular rate cannot be determined but in all the leads there is superimposed notching on various phases of the QRST complex (indicated by x marks) which could be the result of auricular contractions.

sensation in the lower left anterior chest. The offset of these attacks tended to be gradual. Except for a period of weakness of a few days' duration following each bout of tachycardia he felt well between attacks and was able to perform the work of a machinist in civilian life. The family history was negative except for bronchial asthma in one brother.

Physical examination on admission revealed a well developed and well nourished

individual who was 74 inches tall and weighed 170 pounds. His color was good and there was no evidence of any respiratory distress. The temperature was 98.6° F., and the radial pulse was of good quality with a rate of 135 per minute, but irregular. The heart was not enlarged to percussion and there were no thrills or murmurs. The rhythm was grossly irregular and the apical rate was 160 per minute. There was no particular accentuation of any of the heart sounds. The blood pressure was 120 mm. Hg systolic and 75 mm. diastolic. Eyes, ears, nose and throat, lungs, abdomen, extremities and reflexes were all within normal limits. Laboratory studies including complete blood count, urinalysis, blood serologic tests, chest plate and cardiac fluor-

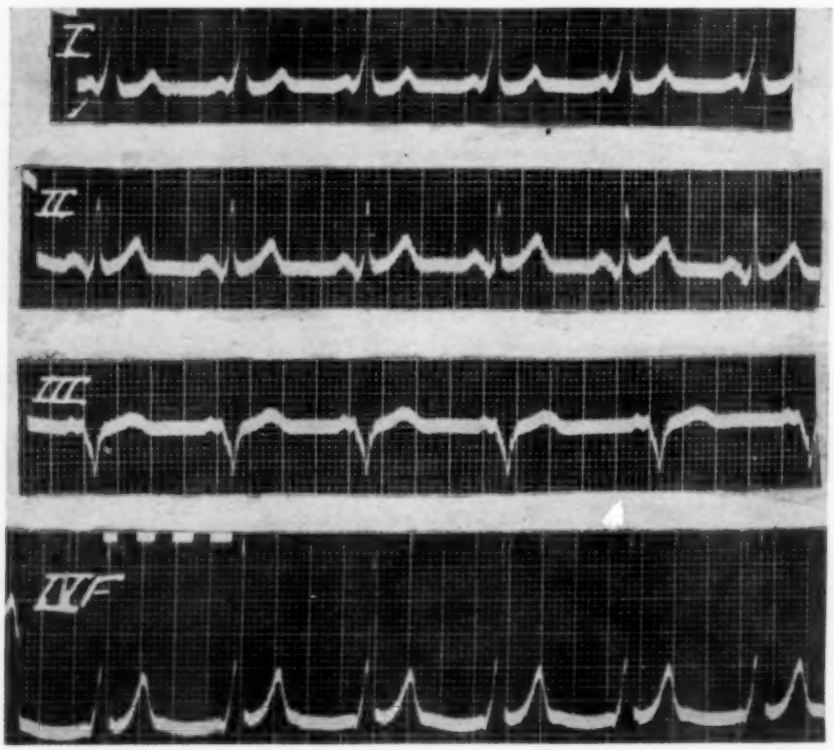

FIG. 2. Electrocardiogram taken on the third hospital day after normal rhythm was established. The auricular and ventricular rate is 78 beats per minute, the PR interval ranges from 0.08 to 0.10 second and the QRS complexes are widened (0.12 second) with initial slurring in all leads.

oscopy were all negative. An allergic survey failed to reveal any significant sensitivity to any of the common pollens, epidermals or foods.

The electrocardiogram on admission revealed an intermittent type of ventricular tachycardia with an occasional supraventricular beat, the ventricular rate ranging between 150 and 160 per minute (figure 1). Subsequent electrocardiograms taken after normal rhythm was established revealed a short PR interval of 0.08–0.10 second and a prolonged QRS complex of 0.12 second with initial slurring in all leads (figure 2).

The patient was placed on quinidine therapy, three grains every three hours, and after a total of 18 grains normal rhythm was established. No evidence of circulatory

or respiratory distress was detected at any time. Quinidine therapy was continued for an additional 24 hours and then discontinued. He remained under observation for a period of six weeks without recurrence of any tachycardia. During this period he was kept ambulatory and participated actively in the hospital reconditioning program without any difficulty. Repeated examination of the heart by several medical officers failed to disclose any abnormalities. The patient was further studied to determine the effect of certain drugs on this syndrome. For five days 4.5 grains of digitalis were given daily and failed to alter the appearance of the electrocardiogram. Atropine sulfate, grains 1/50, given subcutaneously did not alter the PR interval but shortened the duration of the QRS complex to 0.10 second and the complexes assumed a more normal configuration (figure 3). Two c.c. of prostigmine methyl

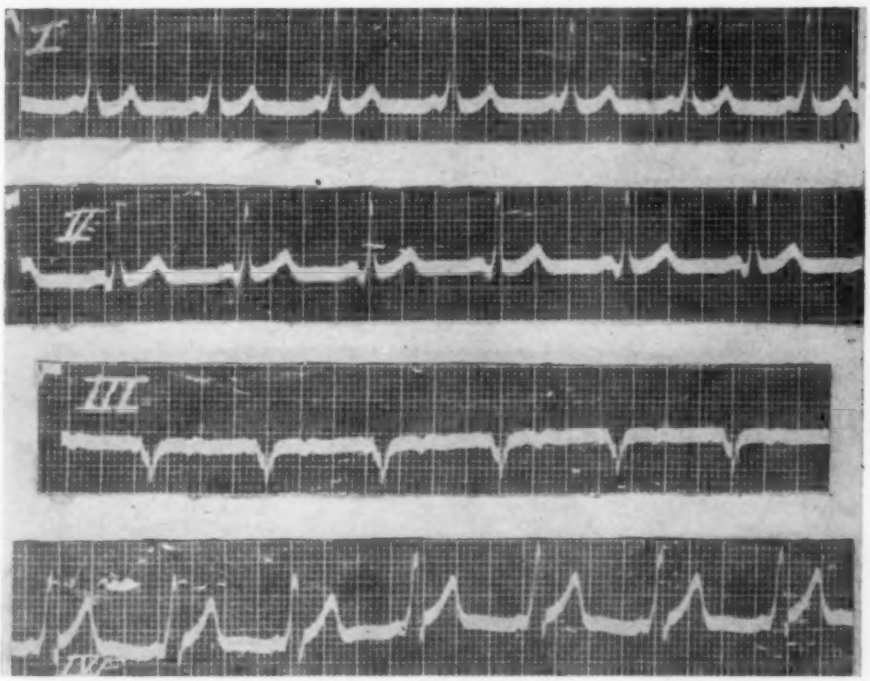

FIG. 3. Electrocardiogram taken 20 minutes after the administration of atropine sulfate grains 1/50 subcutaneously. The significant change is in the duration and the appearance of the QRS complexes. These now measure 0.10 second in duration and the slurring is less marked. The PR interval is unchanged.

salicylate, 1 : 2000 solution given intramuscularly also had no effect on the PR interval but produced suggestive prolongation of the QRS complex to 0.14 second (figure 4). The patient was discharged from the hospital in good condition and symptom free. Owing to the relative infrequency of the recurrent episodes of tachycardia and their relatively benign nature he was not placed on any prophylactic medication.

DISCUSSION

Paroxysmal ventricular tachycardia is an uncommon arrhythmia. It is most often associated with serious underlying organic cardiac disease. Rarely, cases

have been reported in which no organic heart disease could be found.[5, 6] Williams and Ellis [5] divided their series of 36 cases of ventricular tachycardia into two types—the persistent and the intermittent. The latter were characterized by runs of ventricular tachycardia separated by periods of normal rhythm. We feel that the tachycardia seen in this case is of the intermittent type. In figure 1, Leads II, III and IV, there are runs of ventricular tachycardia separated by an occasional supraventricular beat. Furthermore, in this tracing it is possible to detect superimposed notching on various phases of the QRST complex which could represent either an independently beating auricle or retrograde conduction

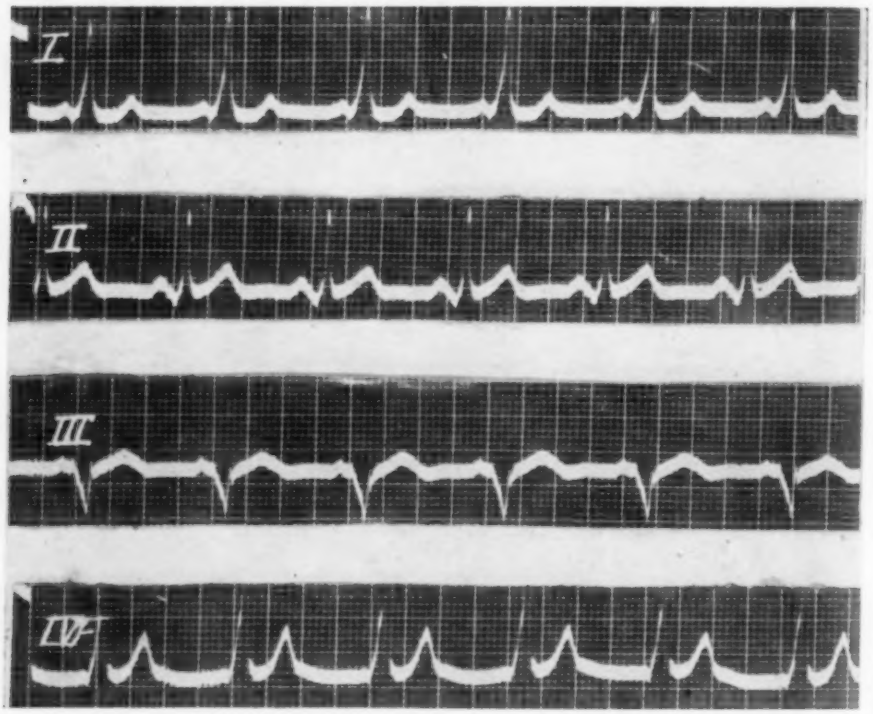

FIG. 4. Electrocardiogram taken 20 minutes after the administration of 2 c.c. of prostigmine methylsalicylate intramuscularly. The QRS complex is somewhat more widened, measuring up to 0.14 second, but is otherwise unaltered. The PR interval is unchanged.

from the ventricle to the auricle. Although a supraventricular tachycardia can produce anomalous ventricular complexes which may simulate those of a ventricular paroxysm, we feel that the tracing obtained in this case more closely resembles that of a true ventricular tachycardia.

This case unquestionably satisfies all the criteria of the Wolff, Parkinson and White syndrome, namely: the short PR interval, the prolonged QRS complexes, initial slurring of the QRS complexes, and paroxysmal attacks of tachycardia. Studies of the effect of various drugs on the electrocardiogram of this patient confirm the results previously reported by other investigators.[7, 8] Atropine shortened the QRS complexes which assumed a more normal contour, whereas

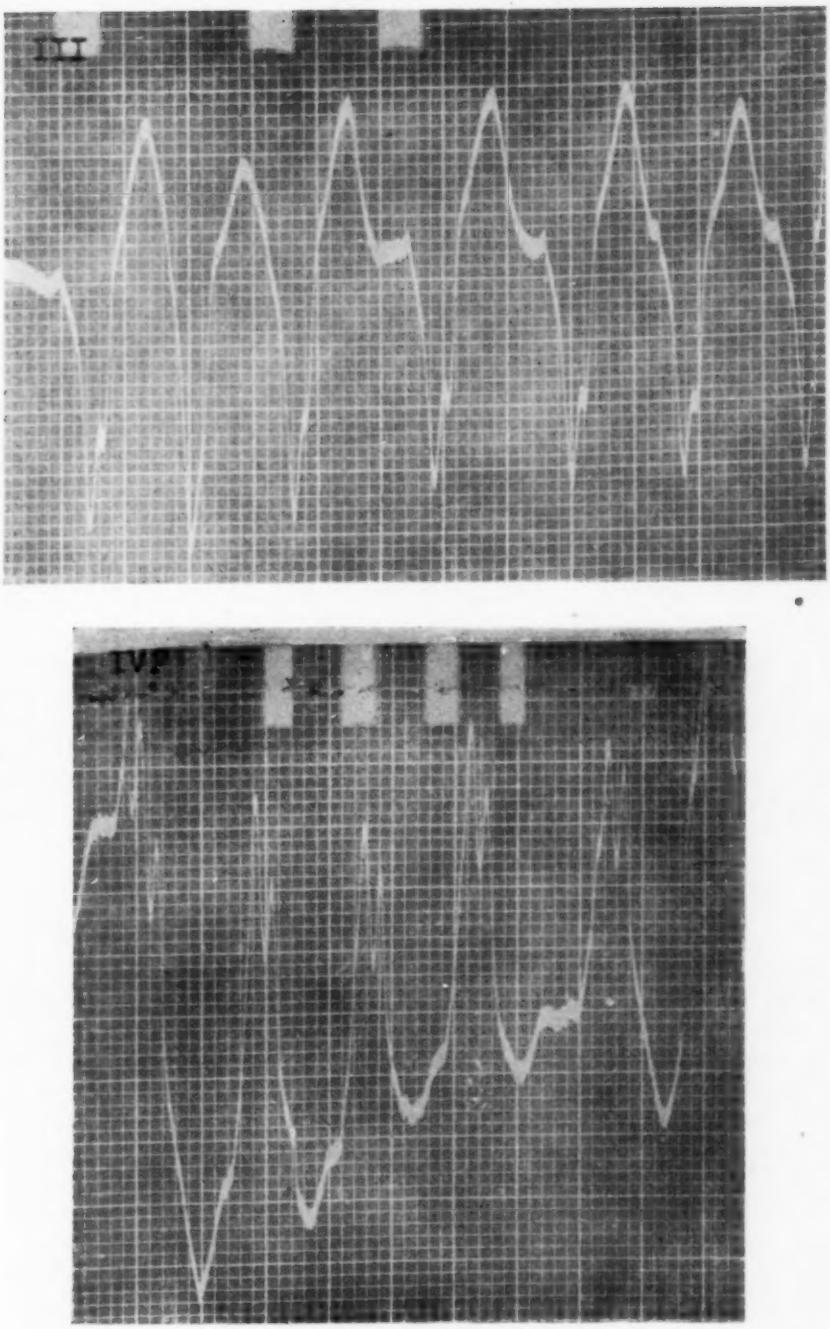

FIG. 5. Portion of electrocardiogram (Leads III and IV F) taken in June 1944 during a paroxysm of tachycardia. This is also a ventricular type of tachycardia and further supports our belief that this arrhythmia in this case is related to the other peculiarities of the conduction system seen in the Wolff-Parkinson-White syndrome.

prostigmine produced widening of these complexes. No changes were noted after digitalis medication, but it is possible that the amount of the drug given may have been insufficient to produce any effect.

The occurrence of paroxysmal ventricular tachycardia and the syndrome of short PR interval and prolonged QRS complex together may be coincidental. As mentioned previously such paroxysms have been reported in structurally normal hearts. In these cases, however, electrocardiograms taken during periods of normal rhythm have usually shown one or more irritable foci in the ventricles as evidenced by ventricular extrasystoles. In the case reported we were unable to demonstrate any structural defect of the heart, and none of the numerous electrocardiograms taken after normal rhythm was established showed any ventricular extrasystoles. Furthermore, none was seen after the patient was given sizeable doses of digitalis. Therefore, we are of the opinion that the paroxysm of ventricular tachycardia seen in this case is not coincidental but is related to the other peculiarities of the conduction system seen in the Wolff-Parkinson-White syndrome (see figure 5). At the present time it is not possible to postulate a mechanism whereby the ventricles can set up a dominant idioventricular rhythm, but it could possibly be related to the theory proposed by Wolferth and Wood [9] as an explanation for the paroxysms of auricular tachycardia seen in this syndrome. These investigators suggested that retrograde conduction from ventricle to auricle through the bundle of Kent could set up the auricular arrhythmia. Butterworth and Poindexter,[10] in 1942, presented an experimental study on cats and dogs which supported this theory. They showed that by the use of an abnormal electrical conducting pathway it was possible to produce typical electrocardiographic tracings with a short PR interval and a prolonged QRS complex. By reversing the flow of electrical stimulation from ventricle to auricle typical paroxysms of auricular tachycardia were produced. The case here reported may lend support to the theory of retrograde conduction from ventricle to auricle. Figure 1 reveals waves superimposed on the QRST complexes which could represent auricular contractions of this nature.

Summary

1. A case of short PR interval with prolonged QRS complex observed during a paroxysm of ventricular tachycardia has been reported, bringing the total of such reported cases to six.

2. The mechanism whereby the ventricle can set up a dominant idioventricular rhythm in this syndrome is unknown, but it could be related to the theory of retrograde conduction from ventricle to auricle through the bundle of Kent.

3. The appearance of the electrocardiogram during the paroxysm of the ventricular tachycardia may lend support to the theory of retrograde conduction as a cause for the paroxysms of tachycardia seen in this syndrome.

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9. Wolferth, C. C., and Wood, F. C.: Further observations on the mechanism of the production of a short PR interval in association with prolongation of the QRS complex, Am. Heart Jr., 1941, xxii, 450.
10. Butterworth, S. J., and Poindexter, C. A.: Short PR interval associated with a prolonged QRS complex, Arch. Int. Med., 1942, lxix, 437.

EDITORIAL

IMMUNIZATION WITH PNEUMOCOCCUS POLYSACCHARIDE

DURING the past thirty years many studies on prophylactic immunization against pneumococcal pneumonia have been made with a number of different antigenic preparations. Among the earliest of these studies was that of Cecil and Austin [1] who tested the effect of prophylactic vaccination against pneumococcal pneumonia at Camp Upton, New York, in 1918. Their vaccine consisted of a saline suspension of heat-killed pneumococci types I, II, and III. Almost all investigators have concluded that immunization exerts a beneficial effect. In most of the studies, however, interpretation of the results was clouded by such variables as differences in the composition of the immunized and control groups; uncertainty as to whether the specific pneumococcal types included in the immunizing preparation were the same as those currently causing pneumonia; failure to determine whether the observed decline in cases in the immunized group was due to a decrease in cases caused by pneumococcal types included in the vaccine; and inadequate control of the antigenicity of the preparations used.

It has been repeatedly demonstrated that animals can be protected against infection by virulent pneumococci by means of antibodies directed against the specific capsular polysaccharides. Francis and Tillett [2] were the first to show that the purified capsular polysaccharides of pneumococci are antigenic for man when injected intracutaneously in a single dose as small as 0.01 milligram. Subsequent immunization projects by various investigators in mental institutions and civilian camps furnished highly suggestive evidence that these polysaccharides may be used successfully to protect human beings against the corresponding types of pneumococcal pneumonia. It remained, however, for MacLeod, Hodges, Heidelberger, and Bernhard [3] to present incontrovertible evidence of the efficacy of such immunization in their perfectly beautiful clinical experiment completed less than one year ago.

The conditions under which MacLeod and his associates carried out their project were as close to ideal as is conceivable. They felt, therefore, that the important sources of error in such a study had been eliminated and that consequently the interpretation of the results could be made with more assurance than had been previously possible. The population chosen was that of a large Army Air Force Technical School, where during the two preceding winters high epidemic rates for pneumococcal pneumonia had prevailed. Information on the serologic types of pneumococci identified from the 1500

[1] CECIL, R. J., and AUSTIN, J. H.: Results of prophylactic inoculation against pneumococcus in 12,519 men, Jr. Exper. Med., 1918, xxviii, 19–41.

[2] FRANCIS, T., JR., and TILLETT, W. S.: Cutaneous reactions in pneumonia; development of antibodies following intradermal injection of type-specific polysaccharide, Jr. Exper. Med., 1930, lii, 573–585.

[3] MACLEOD, C. M., HODGES, R. G., HEIDELBERGER, M., and BERNHARD, W. G.: Prevention of pneumococcal pneumonia by immunization with specific capsular polysaccharides, Jr. Exper. Med., 1945, lxxxii, 445–465.

cases of pneumonia occurring in the first two years was available. Types II, I, V, VII, XII, and IV, in that order, caused 75 per cent of the cases of disease, the rates for the individual types being approximately the same for each of the two years. The living conditions and duties of the population, which had been remarkably uniform during the 1942–43 and 1943–44 seasons, were unchanged during the season 1944–45. A reasonable prediction could thus be made that the incidence of pneumococcal pneumonia during the experimental period would be high and also what types of pneumococci would be involved. Accordingly, the solution used for immunization contained the specific capsular polysaccharides of pneumococcus types I, II, V, and VII; types XII and IV remained as controls. The preparations of polysaccharides were of known antigenic potency as determined previously by inoculation of civilian volunteers.

Immunization by a single subcutaneous injection of the polysaccharides (0.03 to 0.06 milligram of each) was carried out on alternate members of the population, thus insuring a thorough mixing of immunized and non-immunized subjects in all phases of their activities. Computation of the man-days of exposure gave approximately equal values for the two groups. Furthermore, the members of the population could be observed for a reasonably long time, since the students remained at the school for twenty-four weeks. Laboratory facilities were available for typing all cases of pneumonia and, in addition, a continuous carrier survey for pneumococci was carried on, the total sample being over 3700 pharyngeal cultures with an over-all pneumococcal carrier rate of 57.7 per cent.

The evidence obtained by MacLeod and his associates demonstrated clearly that immunization of man with the specific capsular polysaccharides of pneumococcus types I, II, V, and VII is effective in preventing the development of pneumonia due to these types in the immunized subjects. Of equal interest was the observation that immunization of half the population against those four types greatly reduced the incidence of pneumonia due to these types in the non-immunized subjects, the observed incidence of type I, II, V, and VII pneumonia in the non-immunized fraction of the population being but 17.6 per cent of the expected. This conclusion was based on the observed behavior of type XII and type IV in that for each of these types the rates of pneumonia were closely similar for the 1942–43, 1943–44, and 1944–45 seasons.

Earlier studies by other investigators had shown that when pneumococcal pneumonia is epidemic the carrier rates for the epidemic types were high. The same was shown to be true for pneumococcus type XII in MacLeod's study. It seemed probable that the failure of the non-immunized portion of the population to develop high pneumonia rates was due to inhibition of the development of high carrier rates for type I, II, V, and VII as a consequence of its being thoroughly mixed with the immunized portion. In this regard, evidence was presented that the carrier rates for these types in the immunized portion of the population were significantly lower than

in the non-immunized. It was further stressed that the elimination of pneumonia cases in half the population should bring about a comparable elimination of case-contact carriers. For these reasons, it was suggested that the ability of the immunized subjects to carry and disseminate types I, II, V, and VII pneumococci was greatly reduced by specific immunity to these types and that this immune barrier, composed by half the population, greatly reduced the dissemination of these types throughout the whole population.

The time required for the development of immunity following injection of the polysaccharides was believed to be in the neighborhood of two weeks, based on the observation that the only cases of pneumonia among the immunized men that were caused by types I, II, V, or VII developed during the first two weeks after immunization. In support of this conclusion, studies on the serum of immunized individuals showed that specific antibodies developed within this time but usually required three to six weeks to reach their maximum. The duration of immunity was not determined, but it was apparent that six months could be set as a minimum.

Because of the relatively low incidence of pneumococcal pneumonia in civilian populations, antipneumococcal immunization is unlikely to become a general procedure. In certain groups of greater risk such as foundry workers, miners, and inmates of mental institutions, however, immunization would seem to be a desirable procedure. In military populations the greatest incidence of pneumonia occurs in new recruits, so that most benefit would be derived from immunization of this group.

MacLeod and his associates are surely to be congratulated on their carefully controlled piece of clinical research, carried out on a scale sufficiently large to render their conclusions statistically valid. Their study might well be held up as a model for future immunization projects.

W. H. B.

REVIEWS

Physical Chemistry of Cells and Tissues. By RUDOLF HÖBER, University of Pennsylvania School of Medicine, Philadelphia, Pa., with the collaboration of DAVID I. HITCHCOCK, Yale University School of Medicine, New Haven, Conn., J. R. BATEMAN, Mayo Clinic, Rochester, Minn., DAVID R. GODDARD, University of Rochester, Biological Laboratories, Rochester, Minn., and WALLACE O. FENN, University of Rochester, School of Medicine and Dentistry, Rochester, N. Y. The Blakiston Company, Philadelphia. 676 pages; 23.5 × 16 cm. 1945. Price, $9.00.

Physiology is presented from the point of view of physical science in "The Physical Chemistry of Cells and Tissues." In so doing the authors have done much to bridge some of the gaps between some of the concepts of biological and physical science.

The first section consists of a survey of the fundamentals of physical chemistry which presupposes a basic knowledge in this field. In the second section, Dr. Bateman discusses the architectural and functional significance of large molecules in living matter. The contents of the following five chapters are reflected in their titles: Interatomic and Intermolecular Forces; Some Properties of Large Molecules in Solution; Condensed Systems of Large Molecules with Special Reference to the Structure of Fibers; and Some Properties of Films and Membranes.

Dr. Höber has written the next three sections. The first, introductory in nature, presents some of physiochemical properties of protoplasm which are important in the consideration of protoplasm as the basic substance of living matter. Subsequent chapters deal with the properties of the protoplast and the influence of environment (both natural and experimental) upon its permeability and upon its various metabolic activities.

The author is an authority in this field, having spent many years in the study of cell permeability and the effects of environment upon cell potential, permeability to electrolytes, to non electrolytes and upon activity. He has succeeded in correlating much of the significant material and has raised questions which must still be answered. Dr. Höber again takes up the problem of permeability in section 8, following a discussion by Dr. Fenn of the contractility of tissue. This section is concerned primarily with physiological permeability and includes various phases of intestinal absorption and urine formation and the elaboration of digestive secretions.

Dr. Goddard has written an excellent review of respiration in cells and tissue which includes a chapter on respiratory enzymes and the various cycles which may be involved in the stepwise degradation of carbohydrates with the liberation of energy. The volume is an outstanding contribution to biological science.

M. A. A.

Clinical Atlas of Blood Diseases. Sixth Edition. By A. PINEY, M.D., M.R.C.P., and STANLEY WYARD, M.D., F.R.C.P. 138 pages; 20.5 × 13.5 cm. 148 illustrations. 1945. Blakiston Co., Philadelphia. Price, $5.00.

Many people have received help from this little volume. This is the sixth edition. The illustrations are excellent and the text terse and informative. It can be recommended for ready reference.

T. P. S.

BOOKS RECEIVED

Books received during March are acknowledged in the following section. As far as practicable, those of special interest will be selected for review later, but it is not possible to discuss all of them.

Textbook of General Surgery. By WARREN H. COLE, M.D., F.A.C.S., and ROBERT ELMAN, M.D. Fourth Edition. 1118 pages; 24 × 16.5 cm. 1944. D. Appleton-Century Company, New York.

Science and Seizures. By WILLIAM GORDON LENNOX, M.D., Sc. D. Hon. Second Edition. 258 pages; 20 × 12 cm. 1946. Harper & Brothers, New York. Price, $2.00.

Alterações Hepáticas na Tireotoxicose. By P. A. DA COSTA COUTO, Clínico do Instituto dos Bancários. 278 pages; 23.5 × 16 cm. 1944. Borsoi, Rio de Janeiro.

Ambulatory Proctology. By ALFRED J. CANTOR, M.D. 524 pages; 24.5 × 16 cm. 1946. Paul B. Hoeber, Inc., New York. Price, $8.00.

Applied Physiology. By SAMSON WRIGHT, M.D., F.R.C.P. 944 pages; 22.5 × 14.5 cm. 1945. Oxford University Press, New York. Price, $9.00.

The Diagnosis of Nervous Diseases. By Sir JAMES PURVES-STEWART, K.C.M.G., C.B. Ninth Edition. 880 pages; 22 × 14.5 cm. 1945. Williams & Wilkins Company, Baltimore. Price, $11.00.

Skin Diseases in Children. By GEORGE M. MACKEE, M.D., and ANTHONY C. CIPOLLARO, M. D. Second Edition Revised and Enlarged. 448 pages; 24 × 16 cm. Paul B. Hoeber, Inc., New York. Price, $7.50.

Tratado de Cardioangiologia. By PEDRO A. TAPELLA. Docente Libre de Patologia Medica en la Facultad de Buenos Aires. 946 pages; 27 × 18 cm. 1946. Lopez & Etchegoyen S.R.L., Buenos Aires.

Tratado de Patologia Medica. By DR. RODOLFO DASSEN, DR. E. G. FONGI, and DR. O. FUSTINONI. 819 pages; 27 × 18 cm. 1946. Lopez & Etchegoyen S.R.L., Buenos Aires.

Bulletin de L'Académie Suisse des Sciences Médicales. Vol. 1, 1944. FASC 1 & 2. 120 pages. 24 × 16 cm. Benno Schwabe & Co., Basel.

Asma Alergia. By DR. GUIDO RUIZ MORENO. 186 pages; 22.5 × 15 cm. 1946. Lopez & Etchegoyen, S.R.L., Buenos Aires.

Selecões Médicas do Brasil. Fundacão E. Direcão Do Professor Nuno Lisboa. 1945 116 pages; 23 × 16 cm.

COLLEGE NEWS NOTES

New Life Members of the College

The College is gratified to announce the following additional Life Members, listed in the order of subscription:

Dr. Lawrence Arthur Williams, F.A.C.P., Pasadena, Calif.
Dr. Virgil Guy Presson, F.A.C.P., Tucson, Ariz.
Dr. Alfred Winfield Dubbs, F.A.C.P., Allentown, Pa.
Dr. Gertrude Mary Engbring, F.A.C.P., Chicago, Ill.
Dr. Howard Wakefield, F.A.C.P., Chicago, Ill.
Dr. Robert Stanley Flinn, F.A.C.P., Phoenix, Ariz.
Dr. John Joseph Dumphy, F.A.C.P., Worcester, Mass.

Gifts To The College Library

The following gifts of publications by members are gratefully acknowledged.

Lewis H. Bronstein, (Associate), Fort George Meade, Md.—5 reprints
C. T. Burnett, F.A.C.P., Denver, Colo.—1 reprint
Arthur C. Clasen, F.A.C.P., Seattle, Wash.—1 reprint
Norbert Enzer, F.A.C.P., Milwaukee, Wis.—13 reprints
M. B. Guthrie, (Associate), Fort McPherson, Ga.—1 reprint
Sydney Jacobs, F.A.C.P., New Orleans, La.—7 reprints
Arthur L. Kruger, F.A.C.P., Norfolk, Va.—1 reprint
John E. Leach, F.A.C.P., New York, N. Y.—1 reprint
D. O. N. Lindberg, F.A.C.P., Wabasha, Minn.—1 reprint
J. F. McManus, F.A.C.P., Waltham, Mass.—1 reprint
Aaron E. Parsonnet, F.A.C.P., Newark, N. J.—2 reprints
Richard Reeser, Jr., F.A.C.P., St. Petersburg, Fla.—1 reprint
William S. Reveno, F.A.C.P., Detroit, Mich.—2 reprints
H. C. Robinson, F.A.C.P., Grand Rapids, Mich.—1 reprint
J. B. Schwedel, F.A.C.P., New York, N. Y.—10 reprints
Maurice S. Segal, F.A.C.P., Boston, Mass.—1 reprint
Howard Wakefield, F.A.C.P., Chicago, Ill.—1 reprint

The College Headquarters acknowledges with thanks to the author, Dr. Peter J. Steincrohn, F.A.C.P., Hartford, Conn., one copy of his book entitled "Angina Pectoris and Coronary Occlusion," which has been added to the College library.

North Carolina Regional Meeting

The annual regional meeting of the College for the State of North Carolina will be held at the Bowman Gray School of Medicine at Winston-Salem October 18, 1946.

A.C.P. Regional Meeting for Montana and Wyoming

Under the Governorship of Dr. Ernest D. Hitchcock, of Great Falls, Montana, a Regional Meeting of the American College of Physicians was held at Billings, Montana, April 27, 1946, for the states of Montana and Wyoming. In addition to

the program listed below, there was a Business Session and a Dinner at the Northern Hotel.

The program consisted of the following:

Rocky Mountain Spotted Fever in Eastern Montana,
Malcolm D. Winter, M.D., F.A.C.P., Miles City.

Symposium on Chronic Bronchitis and Related Conditions:

1. Perennial Asthma with Emphasis on Intrinsic Asthma,
Maurice A. Shillington, M.D., F.A.C.P., Glendive;
2. Pathogenesis of Pulmonary Fibrosis and Emphysema,
Harold W. Gregg, M.D., F.A.C.P., Butte;
3. Management of the Bronchiectatic Patient,
Keith D. Larson, M.D., Billings (by invitation);
4. Cor Pulmonale,
Ferdinand R. Schemm, M.D., F.A.C.P., Great Falls.

The Murray-Wagner-Dingle Bill,
Allen R. Foss, M.D., F.A.C.P., Missoula.
Rheumatic Fever in Children,
Archie L. Gleason, M.D., F.A.C.P., Great Falls.
Massive Hemorrhage from the Upper Digestive Tract,
W. W. Arrasmith, M.D., F.A.C.P., Casper.

Sufficient time was allotted for discussion of each paper from the floor.

A.C.P. Course In Internal Medicine at the University of California

The Medical Faculty of the University of California will give a course in Internal Medicine at the Medical Center, San Francisco, from June 17–28, inclusive, under the sponsorship of the American College of Physicians and as part of its postgraduate program. Dr. Stacy R. Mettier, Associate Professor of Medicine and Chairman of the Committee on Postgraduate Instruction, will direct the course. Sessions are scheduled daily, five days a week, Monday through Friday, from 9:00 a.m. to 5:00 p.m. Symposia will be given on:

Endocrinology; Goiter; the Anemias; Psychosomatic Relationships; Pulmonary Diseases; Nutrition; Peptic Ulcer; Ulcerative Colitis; Gall Bladder Diseases; Arthritis; Amebiasis; Medical and Obstetrical Problems of Interest to the Internist; Heart Disease; Diseases of the Liver; Neurology; Infectious Diseases; Syphilis; Low Back Pain.

Full details have been published in the postgraduate bulletin of the American College of Physicians. Fees for the course are as follows:

Members of the American College of Physicians ...	$40.00
Non-members	80.00

The American College of Physicians is unable to provide veteran medical officers, members or non-members of the College, the benefits of the amended G. I. Bill of Rights, through which the Veterans Administration pays tuition fees. The College has not the administrative machinery to comply with the regulations through which collections must be made through the Veterans Administration. This course occurs during the two weeks preceding the opening of the American Medical Association meeting in San Francisco. Adequate hotel accommodations have been engaged

through the instrumentality of Dr. Mettier to accommodate all registrants in this course, up to a limit of 150.

For detailed program and application form, address the Educational Director, American College of Physicians, 4200 Pine St., Philadelphia 4, Pa.

A.C.P. Associates Should Attend Annual Sessions of the College

Associates of the College are expected to show an interest in College activities and in the postgraduate facilities offered by the College. Attendance at the Annual Sessions is recorded on the records of every Associate and Fellow of the College. The Credentials Committee anticipates that every Associate, now that the war is over, will attend at least one Annual Session before he comes up for advancement to Fellowship. Associates were excused from this during World War II, first, because many were on military duty; second, because the College had to discontinue its Annual Sessions after 1942.

Dr. Arthur O. Hecker, F.A.C.P., formerly of Harrisburg, Pa., has been appointed Clinical Director of the Veterans Administration Hospital, Coatesville, Pa., effective March 5, 1946.

Dr. Benjamin L. Brock, F.A.C.P., formerly of Waverly Hills Sanatorium, Waverly Hills, Kentucky, has been appointed Clinical Director of the Veterans Administration Hospital, Oteen, N. C.

Dr. Mitchell Bernstein, F.A.C.P., and Dr. Hyman I. Goldstein, Associate, of Camden, N. J., addressed the Northern Medical Association of Philadelphia on March 21, 1946.

Dr. W. P. Anderton, F.A.C.P., New York City, has been appointed Consultant in Medicine to the Flushing Hospital and Dispensary, Flushing, L. I., N. Y.

Dr. Cyril M. MacBryde, F.A.C.P., who has been on the staff of the Washington University School of Medicine since 1933, has moved to Los Angeles and has joined the faculty of the University of Southern California Medical School where he will be Assistant Professor of Clinical Medicine. He will be in charge of the Endocrine Clinic at the Los Angeles County General Hospital. Dr. MacBryde will practice internal medicine in association with the Shelton Clinic, 921 Westwood Boulevard, Los Angeles, and will continue his investigative work on improved forms of insulin and other metabolic and endocrine subjects.

Dr. Francis Gilman Blake, F.A.C.P., Regent of the American College of Physicians, and President of the Army Epidemiological Board, received the Medal of Merit from Major General Norman T. Kirk, F.A.C.P., Surgeon General of the Army, for his outstanding work in planning and organizing the Army Epidemiological Board and as Consultant to the Secretary of War.

Dr. Clarence de la Chapelle, F.A.C.P., Director of the Postgraduate Division of New York University College of Medicine, recently stated that 400 medical veterans have attended postgraduate courses organized by his Division in September 1945.

Dr. William Harvey Perkins, F.A.C.P., Dr. Edward L. Bortz, F.A.C.P., Dr. George Morris Piersol, F.A.C.P., and Dr. William G. Leaman, Jr., F.A.C.P., all of Philadelphia, recently spoke before the Sixteenth Annual Health Institute of the Woman's Auxiliary to the Philadelphia County Medical Society.

Dr. Max Pinner, F.A.C.P., formerly Clinical Professor of Medicine at Columbia University College of Physicians and Surgeons, and Chief of the Division of Pulmonary Diseases, Montefiore Hospital for Chronic Diseases, New York City, has removed to 463 Vermont Ave., Berkeley, Calif., and has discontinued the private practice of medicine. His time will be devoted to the editorship of the American Review of Tuberculosis.

Major Rafael Rodriguez-Molina, Medical Corps, F.A.C.P., has been awarded the Army Commendation Ribbon for commendable service from May 22, 1942 to February 12, 1946 as Assistant Chief and Chief of the Medical Service, 161st General Hospital, A.P.O. 851, U. S. Army.

Dr. Anthony Bassler, F.A.C.P., of New York City has been selected as Vice-President of the International Gastroenterologic Society and President of the permanent International Committee of the organization.

An important forward step in the establishment of professional graduate training for Army doctors has been taken with the enactment of the new Army Regulation 350–1010, which authorizes the establishment of an organized program of graduate education for "the elevation of the general level of professional qualifications of all Medical Corps officers." The Surgeon General assisted by the professional consultants in the various specialties will exercise overall organization and supervision of the program of graduate education in the Army Medical Corps. The objectives are numerous, and include provision for adequate training and qualification of professional and administrative specialists, ample opportunities for professional advancement in clinical and research medicine, establishment of all Army Hospitals as teaching institutions, and finally, to develop the art and science of medicine and to encourage continuous postgraduate medical teaching and education.

This organized program will be carried out in consecutive steps by means of Army interneships, mixed residencies, residencies in medical and surgical subspecialties, postgraduate subspecialty training, and projects in military medical research. Training in the basic medical sciences will be conducted concurrently with other phases of this program.

The chief medical director of the Veterans Administration, Brigadier General Paul R. Hawley, recently announced from Washington the following appointments:

Dr. Charles C. Wolferth, F.A.C.P., University of Pennsylvania Hospital, chief of the cardiology section of the Veterans Administration's Professional Services Division.

Dr. Albert M. Snell, F.A.C.P., Mayo Clinic, chief of the gastro-enterology section.

Brig. Gen. James S. Simmons, F.A.C.P., Boston, Army Medical Corps, chief of tropical medicine.

Dr. Harry L. Alexander, F.A.C.P., Barnes Hospital, St. Louis, chief of allergy.

Dr. Richard A. Kern, F.A.C.P., University of Pennsylvania Hospital, chief consulting internist.

The Nineteenth Graduate Fortnight of the New York Academy of Medicine will be held during the dates October 7 and 18, 1946, on the subject of Tumors. The Fortnight will include Evening Lectures, Morning Panel Discussions, Scientific Exhibits and demonstrations at the Academy; and Afternoon Hospital Clinics at leading hospitals of New York City.

Physicians, who are not Fellows of the Academy, may secure registration by sending name and address, accompanied by check for five dollars, to the Secretary of the Graduate Fortnight Committee, 2 East 103rd Street, New York 29, New York.

Medical Officers of the Army, Navy and United States Public Health Service, on active duty, will be admitted to all sessions without registration fee.

Fellowships for one year of graduate study in health education, leading to a master's degree in public health, are being offered to qualified men and women by the U. S. Public Health Service through funds made available by the National Foundation for Infantile Paralysis, according to a recent release from the Federal Security Agency of the U. S. Public Health Service, Washington 25, D. C.

These fellowships provide a stipend of $100 a month in addition to tuition and travel expenses for the entire period of academic and field training starting in the fall of 1946. Persons accepting fellowships will be expected to work in the field of health education for at least two years after completion of training. Applications may be secured from the Office of the Surgeon General, U. S. Public Health Service, Washington 25, D. C., and should be returned thereto not later than June 1, 1946.

The annual intensive course in Electrocardiography for graduate physicians will be given at Michael Reese Hospital in Chicago from August 19–31, 1946. The Director is Dr. Louis N. Katz, F.A.C.P. Applications should be made to the Cardiovascular Department of the Michael Reese Hospital, 29th Street & Ellis Avenue, Chicago 16, Ill.

Dr. Joseph R. Ridlon, F.A.C.P., a Medical Director in the U. S. Public Health Service, has recently been retired and will be located in Gorham, Maine.

In July 1942 Capt. Robert Gaylord Davis, (MC) USN, F.A.C.P., was reported "missing in action" and for almost the duration of the war was thought to have been dead. However, he was found in a Japanese Prisoner of War Camp and liberated in August 1945 and returned to the United States during September. Following a period of hospitalization and rehabilitation leave, Capt. Davis was assigned again to active duty. However, he is scheduled for detachment to pre-retirement leave and will be officially retired on August 1, 1946.

Capt. Lyle Jay Roberts, (MC) USN, F.A.C.P., was taken prisoner by the Japanese in the early part of the war, but liberated during September 1945 and returned to the United States in October. Following a period of hospitalization, he was granted a rehabilitation leave and has now reported for active duty again.

Dr. James E. Cottrell, F.A.C.P., formerly of Philadelphia, was separated from the Medical Corps of the Army during March 1946, and has accepted a full time position in the Veterans Administration as Chief of the Medical Service in the Veterans Hospital, Memphis, Tenn.

Dr. Chester S. Keefer, F.A.C.P., Boston, and Dr. J. Burns Amberson, Jr., F.A.C.P., New York, recently addressed the Connecticut State Medical Society at its 154th meeting in Hartford, Conn.

Dr. William S. Middleton, F.A.C.P., Regent of the American College of Physicians, Madison, Wis., addressed the 95th session of the Iowa State Medical Society in Des Moines, Iowa.

Dr. Allen W. Cowley, F.A.C.P., Harrisburg, Pa., was recently awarded the Seibert Memorial, given biennially by the Harrisburg Academy of Medicine. This award of $500 is made to a physician under 45 who has "preëminently distinguished himself in his profession and whose life as a citizen and scholar has been broad, unselfish and exemplary."

Dr. Reginald Fitz, F.A.C.P., Regent of the American College of Physicians, and Dr. Dwight O'Hara, F.A.C.P., both of Boston, Mass., spoke before the 50th anniversary dinner of the Tufts Medical Alumni Association on April 10.

Dr. Charles A. Doan, F.A.C.P., Columbus, Ohio, delivered the fifth Edwin R. Kretschmer Memorial Lecture in Chicago on April 26.

Dr. Stockton Kimball, F.A.C.P., Buffalo, N. Y., has been appointed Acting Dean of the University of Buffalo School of Medicine.

Dr. Jean A. Curran, F.A.C.P., Brooklyn, N. Y., was a guest at the annual dinner given by the Medical Society of the County of Kings and the Academy of Medicine of Brooklyn on February 20 in honor of sixteen ex-presidents of this society.

The American Physicians Literary Guild was organized in San Francisco January 2, 1946. All physicians in America who write as an avocation are invited to join the guild. Nonmedical essays, monographs, short stories, or poems will be given competitive consideration by the guild for presentation at a literary exhibition during the annual session of the American Medical Association. Additional information may be obtained through Dr. F. H. Redewill, Secretary, Flood Building, San Francisco 2, Calif.

Dr. Joseph Hughes (Associate), Philadelphia, has been appointed Professor of Psychiatry of the Woman's Medical College of Pennsylvania. At present he holds the rank of commander and is chief of the neuropsychiatric service at the Philadelphia Naval Hospital.

Dr. Albert W. Lapin of Montreal, Canada, has been awarded a Clinical Fellowship in Medicine by the Committee on Fellowships and Awards of the American College of Physicians, beginning May 1, 1946.

Dr. Lapin plans to spend this clinical year at the University Hospital, Ann Arbor, Michigan, doing postgraduate work in Cardiology under Dr. Frank N. Wilson; also at the Massachusetts General Hospital under Dr. Paul D. White, and at Emory University under Dr. E. A. Stead.

Western Michigan Members Organize for Regional Meetings

On March 20, 1946, thirty-six Fellows met at Muskegon, Michigan, to organize a regional group, or chapter, of the American College of Physicians "to promote friendship and understanding among the Fellows and Associates in Western Michigan, and to foster scientific investigation in the field of internal medicine." "The College of Physicians of Western Michigan" was chosen as the name of the group, and membership shall be limited to Fellows and Associates of the American College of Physicians living in or adjacent to that area commonly called Western Michigan. The permanent management of the organization shall be vested in a Secretary who shall be elected at the first meeting of each calendar year by popular vote of those members present. Meetings shall be held three times a year, in the fall, winter and spring seasons, and the Secretary is charged with the responsibility of appointing a chairman of each meeting. The chairman shall arrange a suitable scientific program and make such other arrangements as necessary. The cost of conducting the meetings will be prorated among those in attendance.

Dr. Douglas Donald, Detroit, College Governor for Michigan, was in attendance and addressed the group. Dr. Burton R. Corbus, F.A.C.P., Grand Rapids, acted as Chairman and Dr. William LeFevre, F.A.C.P., Muskegon, was appointed Secretary pro tempore, and was later formally elected Secretary.

Colonel A. R. Gaines, F.A.C.P., of the Percy Jones Hospital, addressed the dinner meeting at the Muskegon Country Club and presented an invitation for the next meeting to be held at the Percy Jones General Hospital, which invitation was accepted.

Bowman Gray School of Medicine of Wake Forest College

The Board of Trustees of Wake Forest College, at a meeting held in Wake Forest on March 26, acted favorably on an offer from the Z. Smith Reynolds Foundation of an endowment fund with a present market value of $10,500,000. The charter of the Foundation provides that 20 per cent of the income be added to the principal until, through this and other contributions, it reaches $50,000,000. The offer stipulated that Wake Forest College be moved to Winston-Salem, where the Bowman Gray School of Medicine of Wake Forest College and the North Carolina Baptist Hospital are now located. Final decision in the matter will be made by the North Carolina Baptist State Convention. Officers and directors of the Z. Smith Reynolds Foundation are: W. N. Reynolds, President; Stratton Coyner, Secretary; Richard J. Reynolds, Mary Reynolds Babcock, Nancy Reynolds Bagley, and W. R. Hubner.

A gift of $125,000 has been recently received from Gordon Gray, son of the late Bowman Gray. The greater portion of the gift will be used to develop a department of Psychiatry.

Dr. Lloyd J. Thompson, formerly a member of the staff of the department of Psychiatry of Yale University School of Medicine, and Chief Consultant in Psychiatry for the European Theatre during the war, has been elected Professor of Psychiatry and director of the department of Neuropsychiatry.

Drs. J. P. Davis, Charles H. Reid, Jr., and Joseph B. Stevens have been appointed to the faculty with the title of Assistant in Medicine.

The fourth Commencement of the Bowman Gray School of Medicine was held on March 24. Forty-two graduates were awarded the degree of Doctor of Medicine. Dr. Thomas T. Mackie of New York delivered the commencement address.

Dr. Wingate M. Johnson of the Department of Medicine spoke at the Annual Clinical Conference of the Chicago Medical Society on March 5. His subject was "The Management of the Patient with Peptic Ulcer."

Dr. George T. Harrell, Jr., of the Department of Medicine, took part in the program of the New Orleans Post-Graduate Medical Assembly, which was held April 1–4.

Dr. Robert P. Morehead of the Department of Pathology has been appointed educational director for the North Carolina division of the Field Army of the American Cancer Society.

Retirements from Service

Since the last publication of this journal, the following members of the College have been reported retired or on terminal leave (to April 12, 1946 inclusive).

Sidney Adler, Detroit, Mich. (Comdr., MC, USNR)
Ralph I. Alford, Montclair, N. J. (Major, MC, AUS)
William H. Algie, Kansas City, Kan. (Comdr., MC, USNR)

John S. Bagwell, Dallas, Tex. (Major, MC, AUS)
Benjamin M. Baker, Jr., Baltimore, Md. (Colonel, MC, AUS)
Fred E. Ball, Chicago, Ill. (Lt. Col., MC, AUS)
Joseph Bank, Phoenix, Ariz. (Lt. Col., MC, AUS)
Lewis Barbato, San Antonio, Tex. (Major, MC, AUS)
Clarke H. Barnacle, Denver, Colo. (Lt. Col., MC, AUS)
Raymond L. Barrett, Springfield, Mass. (Lt. Col., MC, AUS)
Theodore B. Bayles, Boston, Mass. (Lt. Col., MC, AUS)
Morris B. Bender, New York, N. Y. (Comdr., MC, USNR)
Julien E. Benjamin, Cincinnati, Ohio (Colonel, MC, AUS)
Dudley W. Bennett, San Francisco, Calif. (Capt., MC, USNR)
James B. Berardi, Chicago, Ill. (Lt. Col., MC, AUS)
William G. Bernhard, Newark, N. J. (Lt. Col., MC, AUS)
Arthur Bernstein, Chicago, Ill. (Lt., MC, USNR)
Edward G. Billings, Denver, Colo. (Colonel, MC, AUS)
Charles T. Bingham, West Hartford, Conn. (Comdr., MC, USNR)
Benjamin J. Birk, Milwaukee, Wis. (Colonel, MC, AUS)
Belford C. Blaine, Pottsville, Pa. (Major, MC, AUS)
Theodore L. Bliss, Akron, Ohio (Colonel, MC, AUS)
Meyer Bloom, Johnstown, Pa. (Capt., MC, AUS)
Henry A. Bradford, Detroit, Mich. (Capt., MC, AUS)
James A. Bradley, St. Petersburg, Fla. (Lt. Comdr., MC, USNR)
Lewis W. Brown, Newark, N. J. (Comdr., MC, USNR)
Philip W. Brown, Rochester, Minn. (Lt. Col., MC, AUS)
Harvey C. Brownley, Lynchburg, Va. (Lt. Col., MC, AUS)
James G. Bruce, Springfield, Mass. (Major, MC, AUS)
Howard G. Bruenn, New York, N. Y. (Comdr., MC, USNR)
L. Clair Burket, Altoona, Pa. (Major, MC, AUS)

Walter S. Burrage, Boston, Mass. (Capt., MC, USNR)
Hildahl I. Burtness, Santa Barbara, Calif. (Comdr., MC, USNR)
W. Turner Bynum, Chickasha, Okla. (Lt., MC, USNR)

Richard B. Capps, Chicago, Ill. (Lt. Col., MC, AUS)
Charles M. Caravati, Richmond, Va. (Colonel, MC, AUS)
Louis H. Charney, Oklahoma City, Okla. (Lt. Col., MC, AUS)
Eric MacMillan Chew, Seattle, Wash. (Lt. Col., MC, AUS)
Austin B. Chinn, Washington, D. C. (Lt. Col., MC, AUS)
Richard J. Clark, Winchester, Mass. (Major, MC, AUS)
Thomas A. Clawson, Jr., Salt Lake City, Utah (Colonel, MC, AUS)
Charles B. Coggin, Los Angeles, Calif. (Major, MC, AUS)
Ben H. Cooley, Norman, Okla. (Colonel, MC, AUS)
James E. Cottrell, Philadelphia, Pa. (Lt. Col., MC, AUS)
H. Dick Countryman, Rockford, Ill. (Lt. Col., MC, AUS)
George B. Craddock, Lynchburg, Va. (Major, MC, AUS)
Robert M. Craig, Dayton, Ohio (P. A. Surgeon, USPHS(R))
George W. Cramp, Brooklyn, N. Y. (Capt., MC, USNR)
Joseph D. Croft, Evanston, Ill. (Comdr., MC, USNR)
John K. Curtis, New York, N. Y. (Comdr., MC, USNR)
Edward H. Cushing, Cleveland, Ohio (Capt., MC, USNR)

Charles M. Darnall, Austin, Tex. (Major, MC, AUS)
Robert G. Davis, Washington, D. C. (Capt., MC, USN)
Albert M. DeArmond, Indianapolis, Ind. (Lt. Col., MC, AUS)
John K. Deegan, Ithaca, N. Y. (Major, MC, AUS)
Edward A. Delarue, Jr., Richmond, Va. (Major, MC, AUS)
William F. Dobyns, Aspinwall, Pa. (Lt. Col., MC, AUS)
William M. Donohue, Houston, Tex. (Major, MC, AUS)
Frederic G. Dorwart, Muskogee, Okla. (Lt. Col., MC, AUS)
Albert H. Douglas, Jamaica, N. Y. (Comdr., MC, USNR)
Alexander S. Dowling, Corning, N. Y. (Comdr., MC, USNR)
Morris L. Drazin, Jackson Heights, L. I., N. Y. (Lt. Col., MC, AUS)
Thomas J. Dry, Rochester, Minn. (Lt. Col., MC, AUS)
Alfred W. Dubbs, Allentown, Pa. (Major, MC, AUS)
Joseph L. Duffy, London, Ont., Can. (Lt. Col., RCAMC)
Lawrence N. Durgin, Amherst, Mass. (Major, MC, AUS)
Robert B. Durham, Atlantic City, N. J. (Comdr., MC, USNR)

Joseph C. Edwards, St. Louis, Mo. (Lt. Col., MC, AUS)
Joseph C. Ehrlich, Chicago, Ill. (Lt. Col., MC, AUS)
Herbert Eichert, Miami, Fla. (Lt. Comdr., MC, USNR)
Clarence K. Elliott, Lincoln, Nebr. (Comdr., MC, USNR)
F. George Elliott, Edmonton, Alta., Can. (Major, RCAMC)
Ephraim P. Engleman, Boston, Mass. (Major, MC, AUS)
Herbert K. Ensworth, New York, N. Y. (Major, MC, AUS)
Clarence W. Erickson, Pittsburg, Kan. (Capt., MC, AUS)
Richard D. Evans, Beverly Hills, Calif. (Lt. Col., MC, AUS)
David W. Exley, Miami Beach, Fla. (Major, MC, AUS)

Elliston Farrell, New Orleans, La. (Lt. Col., MC, AUS)
Marcus A. Feinstein, New York, N. Y. (Major, MC, AUS)
James O. Finney, Gadsden, Ala. (Major, MC, AUS)
Russell A. Flack, La Fayette, Ind. (Comdr., MC, USNR)
Gerald Flaum, New York, N. Y. (Comdr., MC, USNR)
Charles A. Flood, New York, N. Y. (Lt. Col., MC, AUS)
Maurice P. Foley, Los Angeles, Calif. (Major, MC, AUS)
John V. Fopeano, Kalamazoo, Mich. (Lt. Col., MC, AUS)
Donald E. Forster, Portland, Ore. (Major, MC, AUS)
Frank P. Foster, Boston, Mass. (Lt. Col., MC, AUS)

Leon J. Galinsky, Oakdale, Iowa (Major, MC, AUS)
Clarence L. Gardner, Jr., Aurora, Ill. (Lt. Col., MC, AUS)
Lawrence E. Geeslin, Atlanta, Ga. (Lt. Col., MC, AUS)
Mark L. Gerstle, Jr., San Francisco, Calif. (Capt., MC, USNR)
William T. Gibb, Jr., New York, N. Y. (Comdr., MC, USNR)
Warren M. Gilbert, Rome, Ga. (Lt. Col., MC, AUS)
Samuel M. Gingold, Detroit, Mich. (Major, MC, AUS)
Harold I. Ginsberg, Detroit, Mich. (Capt., MC, AUS)
Daniel A. Glomset, Rochester, Minn. (Capt., MC, AUS)
Jacob S. Golden, Chicago, Ill. (Capt., MC, AUS)
Walter Goldfarb, New York, N. Y. (Lt. Col., MC, AUS)
Bernard A. Goldman, New Orleans, La. (Major, MC, AUS)
Milton J. Goldstein, Scranton, Pa. (Major, MC, AUS)
Robert W. Gordon, Denver, Colo. (Major, MC, AUS)
G. Philip Grabfield, Boston, Mass. (Colonel, MC, AUS)
Robert W. Graham, Ottawa, Ont., Can. (Major, RCAMC)
Edward A. Greco, Portland, Maine (Lt. Col., MC, AUS)
Emil H. Grieco, Bayonne, N. J. (Major, MC, AUS)
William H. Griffith, Los Angeles, Calif. (Major, MC, AUS)
Harold J. Gunderson, Everett, Wash. (Major, MC, AUS)
Lewis Gunther, Los Angeles, Calif. (Comdr., MC, USNR)
Ramsdell Gurney, Buffalo, N. Y. (Major, MC, AUS)

William E. Hall, Meriden, Conn. (Comdr., MC, USNR)
George C. Ham, Charlottesville, Va. (Capt., MC, AUS)
George C. Hamilton, Binghamton, N. Y. (Lt. Col., MC, AUS)
Ian B. Hamilton, Canton, Ohio (Lt. Col., MC, AUS)
Carl F. Hammerstrom, Jamestown, N. Y. (Lt. Col., MC, AUS)
H. Phillip Hampton, Tampa, Fla. (Lt. Col., MC, AUS)
J. Fletcher Hanson, Macon, Ga. (Lt. Col., MC, AUS)
Benedict R. Harris, New Haven, Conn. (Comdr., MC, USNR)
Robert P. Harvey, Limon, Colo. (Lt. Col., MC, AUS)
Frederick K. Herpel, West Palm Beach, Fla. (Lt. Col., MC, AUS)
Ford K. Hick, Chicago, Ill. (Colonel, MC, AUS)
Charles S. Higley, Cleveland, Ohio (Lt. Col., MC, AUS)
Donald A. Hirsch, Chicago, Ill. (Major, MC, AUS)
Joseph H. Hodas, New York, N. Y. (Comdr., MC, USNR)
Carl C. Hoffman, II, Harrisburg, Pa. (Major, MC, AUS)

Arthur A. Holbrook, Milwaukee, Wis. (Lt. Col., MC, AUS)
W. Paul Holbrook, Tucson, Ariz. (Colonel, MC, AUS)
Joseph L. Hollander, Philadelphia, Pa. (Major, MC, AUS)
Thomas N. Horan, Detroit, Mich. (Major, MC, AUS)
Benjamin Horn, Bridgeport, Conn. (Major, MC, AUS)
Allen E. Hussar, New York, N. Y. (Lt. Col., MC, AUS)
Adolph M. Hutter, Fond du Lac, Wis. (Comdr., MC, USNR)

Donald W. Ingham, Washington, D. C. (Lt. Col., MC, AUS)

Louis Jaffe, Detroit, Mich. (Capt., MC, AUS)
Thomas C. Jaleski, New Rochelle, N. Y. (Comdr., MC, USNR)
Edward R. Janjigian, Danville, Pa. (Major, MC, AUS)
Benjamin Jeffries, Detroit, Mich. (S. A. Surgeon, USPHS(R))
William N. Jenkins, Port Gibson, Miss. (Lt. Col., MC, AUS)
Joseph F. Jenovese, Hartford, Conn. (Lt. Comdr., MC, USNR)
Charles A. Jones, Philadelphia, Pa. (Lt. Comdr., MC, USNR)
Robert H. Jordan, New Haven, Conn. (Major, MC, AUS)
Allen I. Josey, Columbia, S. C. (Colonel, MC, AUS)
Irving R. Juster, Glen Falls, N. Y. (Lt. Col., MC, AUS)

John S. Kapernick, Rochester, Minn. (Major, MC, AUS)
George J. Kastlin, Pittsburgh, Pa. (Colonel, MC, AUS)
H. Worley Kendell, Chicago, Ill. (Surgeon, USPHS(R))
Baldwin L. Keyes, Philadelphia, Pa. (Colonel, MC, AUS)
Ernest Q. King, Washington, D. C. (Lt. Col., MC, AUS)
Otis G. King, Bluefield, W. Va. (Major, MC, AUS)
J. Murray Kinsman, Louisville, Ky. (Lt. Col., MC, AUS)
Elmer A. Kleefield, Forest Hills, N. Y. (Major, MC, AUS)
Albert P. Knight, Waverly, N. Y. (Lt. Col., MC, AUS)
George M. Knowles, Hackensack, N. J. (Lt. Col., MC, AUS)
Samuel I. Kooperstein, Jersey City, N. J. (Lt. Col., MC, AUS)

Charles A. Landshof, Jersey City, N. J. (Lt. Col., MC, AUS)
Harry E. Landt, Cincinnati, Ohio (Capt., MC, AUS)
Louis B. Laplace, Philadelphia, Pa. (Colonel, MC, AUS)
William H. Leake, Los Angeles, Calif. (Capt., MC, USNR)
William V. Leary, Rochester, Minn. (Lt. Col., MC, AUS)
Edward P. Leeper, Dallas, Tex. (Lt. Col., MC, AUS)
Charles E. Lemmon, Detroit, Mich. (Lt. Col., MC, AUS)
John B. Levan, Reading, Pa. (Lt. Col., MC, AUS)
Robert C. Levy, Chicago, Ill. (Capt., MC, AUS)
Howard P. Lewis, Portland, Ore. (Colonel, MC, AUS)
Leon Lewis, New York, N. Y. (Comdr., MC, USNR)
Harry R. Lipton, Atlanta, Ga. (Surgeon, USPHS(R))
Joe H. Little, Mobile, Ala. (Lt. Col., MC, AUS)
Leo W. Lloyd, Durango, Colo. (Capt., MC, AUS)
Putnam C. Lloyd, New York, N. Y. (Lt. Col., MC, AUS)
Robert B. Logue, Atlanta, Ga. (Lt. Col., MC, AUS)

Julian S. Long, Wilkes-Barre, Pa. (Major, MC, AUS)
C. Ray Lounsberry, San Diego, Calif. (Capt., MC, USNR)
William S. Love, Jr., Baltimore, Md. (Colonel, MC, AUS)
Eugene L. Lozner, Boston, Mass. (Comdr., MC, USNR)
Clayton J. Lundy, Chicago, Ill. (Major, MC, AUS)
Ralph Lynch, Pittsburgh, Pa. (Lt. Col., MC, AUS)

Willard Machle, Cincinnati, Ohio (Colonel, MC, AUS)
Thomas T. Mackie, New York, N. Y. (Colonel, MC, AUS)
James M. MacMillan, Detroit, Mich. (Capt., MC, AUS)
John E. Manley, Scranton, Pa. (Lt. Col., MC, AUS)
Gilbert H. Marquardt, Chicago, Ill. (Colonel, MC, AUS)
Douglas D. Martin, Tampa, Fla. (Comdr., MC, USNR)
John W. Martin, Jr., Cleveland, Ohio (Lt. Comdr., MC, USNR)
Thomas W. Martin, Pittsburgh, Pa. (Lt. Col., MC, AUS)
Walter P. Martin, Santa Barbara, Calif. (Capt., MC, AUS)
Arthur M. Master, New York, N. Y. (Capt., MC, USNR)
J. Fred Mathers, Orlando, Fla. (Major, MC, AUS)
Marsh McCall, New York, N. Y. (Lt. Col., MC, AUS)
Arthur C. McCarty, Louisville, Ky. (Colonel, MC, AUS)
David W. McCarty, Jr., Longmont, Colo. (Major, MC, AUS)
Thomas C. McCleave, Jr., Oakland, Calif. (Comdr., MC, USNR)
William O. McDonald, St. John, N. B., Can. (Lt. Col., RCAMC)
James W. McElroy, Memphis, Tenn. (Capt., MC, AUS)
Francis J. McEvoy, Royal Oak, Mich. (Comdr., MC, USNR)
Sylvester McGinn, Boston, Mass. (Comdr., MC, USNR)
A. Park McGinty, Atlanta, Ga. (Comdr., MC, USNR)
G. Thomas McKean, Detroit, Mich. (Capt., MC, AUS)
James B. McLester, Birmingham, Ala. (Colonel, MC, AUS)
Ralph E. McLochlin, Little Rock, Ark. (Comdr., MC, USNR)
Delbert H. McNamara, Santa Barbara, Calif. (Lt. Comdr., MC, USNR)
Ronald J. McNamara, Charleston, W. Va. (Comdr., MC, USNR)
James H. McNeill, North Wilkesboro, N. C. (Lt. Comdr., MC, USNR)
Samuel Melamed, New York, N. Y. (Lt. Col., MC, AUS)
Oliver J. Menard, Springfield, Mass. (Lt. Col., MC, AUS)
Harold R. Merwarth, Brooklyn, N. Y. (Capt., MC, USNR)
J. Roscoe Miller, Chicago, Ill. (Comdr., MC, USNR)
Lawrence T. Minish, Jr., Louisville, Ky. (Lt. Col., MC, AUS)
Robert H. Mitchell, Plainview, Tex. (Major, MC, AUS)
Matthew Molitch, Atlantic City, N. J. (Lt. Col., MC, AUS)
Henry A. Monat, Washington, D. C. (Capt., MC, USNR)
Frank T. Moore, Akron, Ohio (Col., MC, AUS)
Philip W. Morgan, Emporia, Kan. (Major, MC, AUS)
Samuel Morrison, Baltimore, Md. (Lt. Col., MC, AUS)
Alvin E. Murphy, Staten Island, N. Y. (Comdr., MC, USNR)
Norman L. Murray, Summit, N. J. (Lt. Col., MC, AUS)

Marshall G. Nims, Denver, Colo. (Major, MC, AUS)
F. Garm Norbury, Jacksonville, Ill. (Col., MC, AUS)

Leo L. Orenstein, New York, N. Y. (Major, MC, AUS)
Bergein M. Overholt, Knoxville, Tenn. (Lt. Col., MC, AUS)
George C. Owen, Oshkosh, Wis. (Lt. Col., MC, AUS)

Christopher Parnall, Jr., Rochester, N. Y. (Lt. Col., MC, AUS)
John W. Parsons, Baltimore, Md. (Comdr., MC, USNR)
Ross Paull, La Jolla, Calif. (Colonel, MC, AUS)
Julius R. Pearson, Miami Beach, Fla. (Major, MC, AUS)
L. Lewis Pennock, Pittsburgh, Pa. (Major, MC, AUS)
Evans W. Pernokis, Chicago, Ill. (Comdr., MC, USNR)
Cornelius C. Perrine, Fair Haven, N. J. (Lt. Comdr., MC, USNR)
Carey M. Peters, Boston, Mass. (Major, MC, AUS)
Michael Peters, Telford, Pa. (Major, MC, AUS)
Frank P. Pignataro, Marlboro, N. J. (Lt. Col., MC, AUS)
Harry H. Pote, Philadelphia, Pa. (Lt. Comdr., MC, USNR)
Frederick C. Potter, Cuyahoga Falls, Ohio (Lt. Col., MC, AUS)
Alvin E. Price, Detroit, Mich. (Lt. Col., MC, AUS)

Frank B. Queen, Chicago, Ill. (Colonel, MC, AUS)
Kenneth E. Quickel, Harrisburg, Pa. (Lt., MC, USNR)
Warren W. Quillian, Coral Gables, Fla. (Comdr., MC, USNR)

Herbert W. Rathe, Waverly, Iowa (Lt. Col., MC, AUS)
Edward P. Reh, St. Louis, Mo. (Major, MC, AUS)
John A. Reisinger, Chevy Chase, Md. (Capt., MC, USNR)
H. Walden Retan, Syracuse, N. Y. (Comdr., MC, USNR)
William F. Rexer, Brooklyn, N. Y. (Major, MC, AUS)
John M. Rice, Watertown, N. Y. (Major, MC, AUS)
Murray L. Rich, Covington, Ky. (Lt. Col., MC, AUS)
Alexander D. Robertson, Willard, Ohio (Major, MC, AUS)
Harold A. Robinson, Detroit, Mich. (Major, MC, AUS)
Edwin J. Rose, Washington, D. C. (Colonel, MC, AUS)
Louis Rosenbaum, New York, N. Y. (Capt., MC, AUS)
Andrew I. Rosenberger, Milwaukee, Wis. (Major, MC, AUS)
Theodore B. Russell, New York, N. Y. (Comdr., MC, USNR)
Benjamin H. Rutledge, Baltimore, Md. (Colonel, MC, AUS)
David I. Rutledge, Boston, Mass. (Major, MC, AUS)
Edward J. Ryan, Cleveland, Ohio (Lt. Comdr., MC, USNR)

Albert C. Santy, New York, N. Y. (Comdr., MC, USNR)
Milton S. Saslaw, Miami, Fla. (Major, MC, AUS)
Newton T. Saxl, New York, N. Y. (Capt., MC, USNR)
Robert L. Schaefer, Detroit, Mich. (Lt. Comdr., MC, USNR)
Edward W. Schoenheit, Asheville, N. C. (Comdr., MC, USNR)
Bernard M. Scholder, Mt. Vernon, N. Y. (Comdr., MC, USNR)
Arthur F. Schultz, Ft. Thomas, Ky. (Major, MC, AUS)
Robert Schwartz, Aspinwall, Pa. (Lt. Col., MC, AUS)
George X. Schwemlein, Cincinnati, Ohio (Capt., USPHS(R))
Edward V. Sexton, Teaneck, N. J. (Lt. Comdr., MC, USNR)

Edward G. Seybold, Jackson, Mich. (Capt., MC, AUS)
Louis B. Shapiro, Manteno, Ill. (Lt. Col., MC, AUS)
Samuel A. Shelburne, Dallas, Tex. (Capt., MC, USNR)
John McFarland Sheldon, Ann Arbor, Mich. (Colonel, MC, AUS)
Karl Shepard, High Point, N. C. (Capt., MC, AUS)
Kenneth K. Sherwood, Seattle, Wash. (Major, MC, AUS)
Leonard B. Shpiner, Boston, Mass. (Major, MC, AUS)
Walter M. Simpson, Dayton, Ohio (Capt., MC, USNR)
Elmer R. Smith, Ancon, C. Z. (Lt. Col., MC, AUS)
Lucian A. Smith, Rochester, Minn. (Major, MC, AUS)
Wilson F. Smith, Hartford, Conn. (Lt. Col., MC, AUS)
Albert M. Snell, Rochester, Minn. (Capt., MC, USNR)
Edward D. Spalding, Grosse Pointe Farms, Mich. (Lt. Col., MC, AUS)
Russell J. Spivey, Indianapolis, Ind. (Major, MC, AUS)
Aaron A. Sprong, Sterling, Kan. (Major, MC, AUS)
John S. Staneslow, Waterbury, Conn. (Lt. Comdr., MC, USNR)
Richard P. Stetson, Boston, Mass. (Lt. Col., MC, AUS)
Russell A. Stevens, Wilkes-Barre, Pa. (Lt. Comdr., MC, USNR)
Gilbert M. Stevenson, Ancon, C. Z. (Major, MC, AUS)
Sloan G. Stewart, Atlantic City, N. J. (Colonel, MC, AUS)
Andrew B. Stockton, San Francisco, Calif. (Comdr., MC, USNR)
Charles F. Stone, Jr., Atlanta, Ga. (Capt., MC, AUS)
William E. Storey, Columbus, Ga. (Major, MC, AUS)
Cyrus W. Strickler, Jr., Atlanta, Ga. (Major, MC, AUS)
William J. Sullivan, Bronxville, N. Y. (Lt. Comdr., MC, USNR)
Horatio B. Sweetser, Jr., Minneapolis, Minn. (Capt., MC, USNR)
Ralph E. Swope, New York, N. Y. (Major, MC, AUS)

Henry M. Tabachnick, Portland, Maine (Major, MC, AUS)
R. Henry Temple, Kinston, N. C. (Major, MC, AUS)
Lyndon H. Thatcher, Poughkeepsie, N. Y. (Major, MC, AUS)
Charles M. Thompson, Philadelphia, Pa. (Comdr., MC, USNR)
Jan H. Tillisch, Rochester, Minn. (Major, MC, AUS)
Elam C. Toone, Jr., Richmond, Va. (Lt. Col., MC, AUS)
James H. Townsend, Boston, Mass. (Lt. Col., MC, AUS)
John W. Trenis, Washington, D. C. (Major, MC, AUS)
William H. Trimble, Atlanta, Ga. (Lt. Col., MC, AUS)
Arthur M. Tunick, New York, N. Y. (Lt. Col., MC, AUS)
Arthur R. Twiss, Oakland, Calif. (Major, MC, AUS)

Harold L. Vyner, Brentwood, N. Y. (Major, MC, AUS)

Don C. Wakeman, Topeka, Kan. (Lt. Col., MC, AUS)
Joe Edmund Walker, Long Beach, Calif. (Capt., MC, USNR)
Emmett D. Wall, Chicago, Ill. (Lt. Col., MC, AUS)
Albert W. Wallace, Miami Beach, Fla. (Colonel, MC, AUS)
C. Stewart Wallace, Ithaca, N. Y. (Comdr., MC, USNR)
E. Richmond Ware, Los Angeles, Calif. (Lt. Col., MC, AUS)
Leon H. Warren, Philadelphia, Pa. (Lt. Col., MC, AUS)

Harry Warshawsky, West Lebanon, N. H. (Lt. Col., MC, AUS)
Richard N. Washburn, Rensselaer, Ind. (Lt. Col., MC, AUS)
Sydney P. Waud, Chicago, Ill. (Lt. Col., MC, AUS)
Solomon C. Werch, Chicago, Ill. (Capt., MC, AUS)
George K. Wever, Stockton, Calif. (Major, MC, AUS)
R. James Wharton, Johnson City, N. Y. (Colonel, MC, AUS)
John C. White, New Britain, Conn. (Comdr., MC, USNR)
Benjamin V. White, Hartford, Conn. (Lt., MC, USNR)
Roger S. Whitney, Colorado Springs, Colo. (Major, MC, AUS)
Dwight L. Wilbur, San Francisco, Calif. (Comdr., MC, USNR)
John H. Willard, Philadelphia, Pa. (Comdr., MC, USNR)
Olin G. Wilson, Canton, Ohio (Major, MC, AUS)
William H. Windley, Washington, N. C. (Major, MC, AUS)
Alfred M. Wolfe, Denver, Colo. (Comdr., MC, USNR)
Bernard P. Wolff, Atlanta, Ga. (Lt. Col., MC, AUS)
Bertrand O. Woods, Portland, Ore. (Colonel, MC, AUS)
Robert M. Woods, Milwaukee, Wis. (Capt., MC, AUS)
Jackson W. Wright, Cincinnati, Ohio (Major, MC, AUS)

Asher Yaguda, Newark, N. J. (Comdr., MC, USNR)

Selection of Dr. Ernest W. Goodpasture, Professor of Pathology and Dean of the School of Medicine of Vanderbilt University, Nashville, Tennessee, as the 1946 recipient of the Passano Foundation Award has been announced by the Board of Directors of the Foundation. Presentation of the $5000 cash award will be made at an appropriate ceremony in historic Osler Hall of the Medical and Chirurgical Faculty of Maryland, in Baltimore, on the night of May 15.

The Foundation, which was established in 1944 by the Williams and Wilkins Company, Medical Publishers, of Baltimore, proposes to aid in any way possible the advancement of medical research, especially research that bears promise of clinical application. For the encouragement of such research the Foundation has established the award as one of its activities.

Dr. Emil Novak, Associate in Gynecology in the Johns Hopkins University Medical School; Dr. Nicholson J. Eastman, Professor of Obstetrics in the Johns Hopkins University Medical School; Dr. George W. Corner, Director of the Embryological Laboratory of the Carnegie Institution of Washington, represent the medical profession on the Board of Directors of The Foundation.

Following the presentation of the award by Mr. Edward B. Passano, Chairman of the Board of The Williams & Wilkins Company, Dr. Goodpasture will deliver an address entitled, "Research and Medical Practice."

Dr. Goodpasture receives the award for his original development of the method for propagation of viruses in pure culture by inoculation of chick embryos and for his outstanding contributions to advancement of knowledge of the cell-parasite relationship in bacterial and virus infection.

Prior to Dr. Goodpasture's development of the chick embryo method of propagation of viruses in pure culture, medical research was halted in attempts to study diseases caused by viruses because of the fact that viruses will not multiply in culture media as do bacteria. Therefore virus cultures could not be made available for research into the mechanism of their reactions.

As a result of Dr. Goodpasture's original discovery some ten years ago our knowledge of virus diseases has been immeasurably advanced. New viruses have been identified, the mechanism of virus reactions in the host has been studied and means of protection against many virus diseases have been made possible. Vaccines against several diseases of both man and animal against which little if any protection existed before have now been developed. Defenses against such diseases as fowlpox, smallpox, yellow fever, influenza and typhus fever have been considerably advanced by chick embryo study methods.

OBITUARIES

DR. RALPH CHARLES MATSON

Dr. Ralph Charles Matson, F.A.C.P., Portland, Oregon, passed away on October 26, 1945.

Born January 21, 1880, Dr. Matson received his medical degree from the University of Oregon School of Medicine in 1902. He had extensive postgraduate study at St. Mary's Hospital, London; University of Vienna; University of Berlin; Academy of Medicine, Düsseldorf, Germany; the Brauer Clinic of Hamburg, Germany; and the Victoria Park and Brompton Hospitals of London.

Dr. Matson was head of the Department of Bacteriology at his Alma Mater, later, Associate Clinical Professor of Medicine and Associate Clinical Professor of Surgery, where he had been a member of the Executive Faculty and Co-Director of the Tuberculosis Clinic. He served during World War I as Chief of the Medical Staff, General Hospital No. 21, now the Fitzsimons General Hospital, Denver; Chest Consultant, Multnomah County Hospital and Veterans Administration Facility at Portland. He was the Director of the Department of Thoracic Surgery, Portland Open Air Sanatorium; Visiting Physician, Good Samaritan Hospital and member of the Associate Medical Advisory Board of the National Jewish Hospital of Denver and honorary member of the Staff of the Lymanhurst School for Tuberculosis, Minneapolis; Chairman, Editorial Board, "Diseases of the Chest," and member of the Editorial Board, "Western Journal of Surgery, Obstetrics and Gynecology." He was also Chief Surgeon of the University State Tuberculosis Hospital.

Dr. Matson was also author of numerous articles in English, French and German, dealing with the diagnosis, medical and surgical treatment of pulmonary, tuberculosis, and contributor of sections of several books.

He was a member of the Portland Academy of Medicine, Pacific Interurban Clinical Club, Pacific Coast Surgical Association, American Association for Thoracic Surgery, Oregon State Tuberculosis Society, American Clinical and Climatological Association and American Trudeau Society. He was an honorary member of the Hollywood Academy of Medicine, Minneapolis Surgical Society, Kansas City Southwest Clinical Society and Eastern Oregon District Medical Society. In 1938, he was Vice-Chairman of Thoracic Section, Pan-Pacific Medical Association; Past President, American Sanatorium Association; Past President and member of the Board of Regents of the American College of Chest Physicians; former Vice-President and Director of the National Tuberculosis Association. He was a Fellow of the American College of Surgeons and International College of Surgeons; also a Fellow of the American College of Physicians since 1917.

DR. GEORGE JESSE WRIGHT

Dr. George Jesse Wright, F.A.C.P., Pittsburgh, Pennsylvania, died October 1, 1945, in the Toronto General Hospital, Toronto, Ontario, Canada, at the age of 65, of nephritis.

Born in Pittsburgh, June 1, 1880, Dr. Wright received his A.B. Degree from Harvard University in 1900 and the degree of Doctor of Medicine from the University of Pennsylvania School of Medicine in 1904. His postgraduate appointments were served at the Pennsylvania Hospital in Philadelphia, the Neurological Institute in New York and the Boston Psychopathic Hospital. He was a diplomate of the American Board of Psychiatry and Neurology and member of the American Neurological Association, the American Psychiatric Association and the Association for Research in Nervous and Mental Diseases. For many years, Dr. Wright was Professor of Neurology at the University of Pittsburgh School of Medicine. He became a Fellow of the American College of Physicians in 1931.

Formerly President of the Pennsylvania Psychiatric Society, Dr. Wright was Neurologist to the St. Margaret's Memorial, Mercy, St. Francis and Allegheny General Hospitals in Pittsburgh. He also served as Visiting Neurologist at St. Joseph's Hospital and Dispensary.

His professional services will be sorely missed by his colleagues in western Pennsylvania. The American College of Physicians has lost an outstanding Fellow.

DR. WILLIAM WADDLE RICHARDSON

Dr. William Waddle Richardson, F.A.C.P., died at the Mercer Sanitarium, Mercer, Pa., on June 10, 1945, at the age of 67, of cerebral hemorrhage.

Born in Athens, Ohio, October 8, 1877, Dr. Richardson received his Medical Degree from the University of Pennsylvania School of Medicine in 1902. He pursued postgraduate study in Neurology and Psychiatry at the Psychiatric Clinic, Munich, at the Harvard Medical School, and at the New York Neurological Institute. He received his basic intern training at the Philadelphia General Hospital, and served as Chief Physician to the Norristown (Pa.) State Hospital. For many years he was Medical Director of the Mercer Sanitarium, Mercer, Pa., and Consulting Neuropsychiatrist to the Christian H. Buhl Hospital in Sharon, Pa. He was a member of Beta Theta Pi, Phi Beta Kappa, and Sigma Xi.

Dr. Richardson was a diplomate of the American Board of Psychiatry and Neurology, and was twice elected President of the Mercer County Medical Society. He was a member of the Pennsylvania State Medical Society, the Pittsburgh Neurological Society, the Association for Research in Nervous and Mental Diseases, and also a member of the Central Neuropsychiatric Association.

During World War I, he served in France with the rank of Major in the Medical Corps as Neurologist with Base Hospital No. 11. During World War II, he was Chairman of the Medical Advisory Board No. 10 of the Pennsylvania Selective Service. He became a Fellow of the American College of Physicians in 1923.

DR. WILLIAM VIRGIL WATSON

William Virgil Watson, M.B., F.A.C.P., Toronto, Ontario, Canada, passed away on October 20, 1945.

Born October 31, 1886, Dr. Watson received his M.B. Degree from the University of Toronto Faculty of Medicine in 1914. He served his internship at the Toronto General Hospital, and for many years was a Demonstrator in Therapeutics at his Alma Mater. His private practice was limited to Internal Medicine and Metabolic Diseases. He was author of several published articles which reflected his grasp of Internal Medicine and Metabolism.

Dr. Watson was a member of the Toronto Academy of Medicine, the Ontario Medical Association, Canadian Medical Association and the American Association for the Advancement of Science. He became a Fellow of the American College of Physicians in 1931.

DR. THOMAS GOTTHART JENNY

Dr. Thomas Gotthart Jenny, Associate, died in Miami, Florida on August 31, 1945 at the age of 55, of coronary heart disease and nephritis.

Born September 12, 1885, Dr. Jenny received his medical degree from the University of Pittsburgh School of Medicine in 1907. He limited his practice to Internal Medicine and Cardiology, and was a member of the staff of Western Pennsylvania Hospital. He was also a member of the Allegheny County Medical Society and the Pennsylvania State Medical Association. He was a Fellow of the American Medical Association and had been an Associate of the American College of Physicians since 1924 by virtue of former membership in the American Congress on Internal Medicine.

His active professional life was spent in Pittsburgh, but he retired a year or two ago and moved to Miami, Florida.

DR. CLIFFORD DAVID MERCER

Clifford David Mercer, M.D., F.A.C.P., died December 25, 1945, after an illness of five years with coronary occlusion and complications. Dr. Mercer was born at Addison, Michigan, 1884; he attended the University of Michigan Medical School, 1904–1906; received his M.D. degree, 1908, from Northwestern University Medical School. He served as Resident Physician at the Uniontown Hospital (Pennsylvania), 1908–1909. For many

years he was a member of the staff of the West Union Community Hospital, Commissioner of Insanity, Fayette County, served as Internist on Medical Advisory Board No. 1, Iowa Selective Service, during World War II. He was former President of the Fayette County Medical Society, member of the Iowa State Medical Society, American Medical Association, Iowa Public Health Association; Fellow of the American College of Physicians since 1927 and recently a Life Member.

Dr. Mercer was a quiet, earnest conscientious physician, well loved in his community and respected by his medical colleagues throughout the State.

B. F. Wolverton, M.D., F.A.C.P.,
Governor for Iowa

DR. MAXIMILIAN A. RAMIREZ

Dr. Maximilian A. Ramirez, F.A.C.P., of New York City died on March 4, 1946. Dr. Ramirez was born in Cuba in 1890. He had been a Fellow of the College since 1924. He was educated in the University and Bellevue Medical School and since that time had been very closely connected with the New York Polyclinic Medical School and Hospital. At the time of his death he was Professor of Medicine and Attending Physician at the Polyclinic Medical School and Hospital. He also was Visiting Physician and Director of the Second Medical Division, French Hospital; was director of the Department of Immunology in the French Hospital; Visiting Physician City Hospital; Visiting Physician, Otisville Municipal Sanatorium; Consulting Physician St. Francis Hospital, Poughkeepsie; Consulting Physician, St. Agnes Hospital, White Plains; Consulting Physician, St. Clare's Hospital; Consulting Immunologist Broad Street Hospital.

He served as a first lieutenant in the Medical Corps of the Army in the first World War, was made a Chevalier of the Legion of Honor in 1933, and was presented with a plaque by the Honor Legion of the New York Police Department for his work as honorary consulting police surgeon.

He was a member of the New York Academy of Medicine, and Past President and Trustee of the Medical Society of the County of New York.

He was the author of numerous published papers, and his many friends will mourn the passing of a distinguished doctor.

Asa L. Lincoln, M.D., F.A.C.P.,
Governor for Eastern New York

DR. EDWIN LESLIE GARDNER

"The life of the dead consists in being present in the minds of the living."
—Cicero.

Edwin Leslie Gardner, B.S., M.D., F.A.C.P., Minneapolis, Minnesota, died at his home, January 29, 1946, at the age of 59 years, following a two-year illness. Dr. Gardner, son of William C. and Eva Gardner, was born

August 2, 1886, in Jacksonville, Illinois. He received his high school education at Belmont Military Academy, Belmont, California; entered the University of Minnesota in 1906, and after two years in the College of Science, Literature and Arts, entered the Medical School and was graduated in 1912. He was a member of Nu Sigma Nu and Alpha Omega Alpha. He held first rank in a class of thirty, and was awarded the Bell prize in physical diagnosis. He served an interneship at the Elliot Memorial Hospital and subsequently became assistant to Dr. J. W. Bell, after which he started his career in his chosen specialty, internal medicine.

His society memberships included: Hennepin County Medical Society, (President 1930); Minnesota State Medical Society, (Editing and Publishing Committee of Minnesota Medicine); American Medical Association; Minnesota Pathological Society (President); Minneapolis Clinical Club (President); Minnesota Academy of Medicine (Secretary); Minnesota Society of Internal Medicine (President); American College of Physicians. He was a member of the faculty of the University of Minnesota Medical School, and was attending physician at the Minneapolis General Hospital from 1914 to 1926.

At various times he was on the staff of Glen Lake Sanatorium, St. Mary's, Eitel, Asbury and Northwestern Hospitals. He was associated with Drs. L. S. Ylvisaker, Robert Kennecott, Paul Rowe, Lewis Daniels, Willard White, Charles A. Hallberg, R. S. Ylvisaker and the writer. He was a member of Hennepin Avenue Methodist Church, the University Club and the Professional Men's Club.

He was much the same, both as an undergraduate and as a practitioner, always serious minded and with a singleness of purpose—had he been born a few decades earlier he would have been known as a pioneer, possessing that industry and diligence characteristic of the self-made man. His secret of life was work. It was a habit so ingrained that it brought him the respect of his teachers and confreres, and a large clientele. When later he took up the avocations of photography and music he followed these in the same arduous way that he applied to medicine. In fact, it is more than likely that this not unmixed virtue contributed to his early demise.

T. A. PEPPARD, M.D.

DR. O. F. GOBER

On January 26, 1946, Dr. Olin Farris Gober, F.A.C.P., of Temple, Texas, died suddenly from a heart attack while on duty at Temple Hospital.

Dr. Gober was born in Jackson County, Georgia, on April 4, 1878, being educated in Texas, graduating from the University of Texas Medical Department in 1905. From 1918 he was Chief Physician of the Gulf, Colorado and Santa Fe Railroad, and was head of their Medical Service in Texas. Since 1926 Dr. Gober has been a Fellow of the American College of Physicians.

Spending his entire medical life in Temple, Texas, Dr. Gober was an active member of the staff of Scott and White Clinic, in addition to his large industrial practice. During his forty years of practice in Texas, he was an active worker in both medical and civic affairs, giving freely of his time and counsel when called upon. His kindly personality, understanding nature, and deep sincerity of purpose made him universally loved by everyone who knew him. He is survived by his son, Dr. Olin B. Gober, of Temple, Texas, his wife having died in 1944.

Dr. Gober's passing will be mourned by a host of friends throughout the Southwest, who will remember him always as an ideal physician who never spared himself when there was a service to be performed.

M. D. Levy, F.A.C.P.,
Governor for Texas